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# LXXIII.—CUTANEOUS TORULOSIS

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The incidence of cutaneous torulosis, both in the United States and in Europe, has not as yet reached a point alarming to the physician and the layman. This disease is rare—at least its occurrence has rarely been reported in the literature—and needs closer attention than has heretofore been paid to it with regard to differential diagnosis. The infection usually manifests itself on the skin, as pointed out by Wile,[1] in the form of acneform pustules, granuloma-like ulcers, deep-seated abscesses and nodules, the last-mentioned lesions varying in size and showing no tendency to ulceration.

Clinically the condition is worthy of a great deal of investigation, chiefly because it may simulate closely the lesions of blastomycosis and coccidioidal granuloma, particularly the European blastomycosis of Busse and Buschke. The correct differential diagnosis of these conflicting infections depends usually on the determination of the invading organism by the methods described by Weidman.[2] Such laboratory procedure, however, must be carried out by a person well trained in mycology. A number of yeastlike fungi may be found in cutaneous

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Studies, observations and reports on cutaneous aspects from the Barnard Free Skin and Cancer Hospital, and the School of Medicine, Washington University, Department of Dermatology and Syphilology, Service of Dr. M. F. Engman and Dr. W. H. Mook.

* This paper was intended for publication by Dr. William H. Mook before his death, which occurred on Nov. 3, 1934. Owing to the complications of routine and investigative work that was in progress the material was set aside for such a time as would permit its complete preparation. After Dr. Mook's untimely death the data seemed of sufficient interest to warrant the completion and publication of the report.

1. Wile, U. J.: Cutaneous Torulosis, Arch. Dermat. & Syph. **31**:58 (Jan.) 1935.

2. Weidman, F. D.: Cutaneous Torulosis, South. M. J. **26**:851, 1933.

lesions which under satisfactory conditions may present a picture analogous to that demonstrated for the organism causing torulosis. This is particularly evident in infections produced by some of the imperfect yeasts, which are often confused with Cryptococcus and Zymonema (Endomyces). The response of tissue to these invading microbes varies considerably, but the fungus itself may, and usually does, develop a mucoid capsule around itself, which is demonstrated in india ink preparations as a clear, gelatinous sheath or envelop. The thickness of this so-called capsule varies, but it rarely, if ever, reaches the proportions found in Cryptococcus histolyticus. This feature serves best to differentiate true torulosis from many of the diseases due to contaminating yeastlike organisms.

It is unfortunate that the term torulosis has been completely adopted by the medical profession to indicate a particular infection caused by a yeastlike organism which apparently is misnamed. The generic term Torula as applied to the causative agent of this disease is incorrect and should be replaced by the term Cryptococcus, as will be pointed out in this paper. For the sake of nomenclatorial correctness and with the hope that the term C. histolyticus will come into universal usage, this term is used throughout this paper.

Torulosis as a clinical entity was first believed to be confined to the internal organs, particularly the brain and the spinal cord. As such it has received a great deal of attention in extensive monographs, the first of which was published by Stoddard and Cutler,[3] which included a clinical review and experimental and cultural data with regard to the fungus. The authors stressed the absence of cutaneous involvement. The most recent review in monographic form is that by Freeman.[4] This work is a fine treatise on the histopathologic features of the disease.

The known cases of supposed torulosis of the skin have been reviewed by Weidman [2] and Wile,[1] but brief mention of them will be made here. The first case of cutaneous torulosis was reported by Rappaport and Kaplan [5] as having occurred in a patient who died of generalized torulosis. The disease had involved the cerebrospinal system, lungs and other internal organs. The skin showed symptoms later, in the form of an acneform pustule on the forehead. Cultures of C. histolyticus were grown from these lesions. The cutaneous manifestations were found histologically to be located in the corium and to consist of infiltrations of fibroblasts, round cells, many organisms and a generalized

3. Stoddard, J. L., and Cutler, E. C.: Torula Infection in Man, Monograph 6, Rockefeller Institute for Medical Research, 1916, p. 1.

4. Freeman, W.: Torula Infection of the Central Nervous System, J. f. Psychol. u. Neurol. **43**:236, 1931.

5. Rappaport, B. Z., and Kaplan, B.: Generalized Torula Mycosis, Arch. Path. **1**:720 (May) 1926.

chronic reaction. This picture simulates closely that seen in the case reported in this article.

A doubtful case of torulosis of the skin is that reported by Versé.[6] In this case, there appeared on the body of the patient acneform lesions which later degenerated into ulcers. The patient died of meningitis, and a diagnosis of systemic blastomycosis was made. Autopsy showed a possible generalized torulosis. This case, however, cannot be definitely considered as one of cutaneous torulosis, as concrete evidence was lacking.

In the same year that Rappaport and Kaplan reported their case, McGehee and Michelson [7] reported an infection occurring in a Negress. The patient had a localized abscess, with pain and swelling, in the left groin. Cultures of C. histolyticus were grown from this lesion. There were no systemic or generalized symptoms, and the patient recovered. It is possible that the organism which answered the description of Cryptococcus may have been a weak strain. On the other hand, one cannot rule out the possibility that a saprophytic strain of Cryptococcus, so common on the skin, may enter into a small lesion and there live on débris and multiply rapidly, causing pain and swelling.

The most outstanding case of supposed cutaneous torulosis is that reported by Urbach and Zach.[8] The disease had its origin in the mouth. The lesions spread to the upper jaw and, approximately one year later, to the abdomen, lungs, thigh and neck. The left jaw showed firm, indolent lymph nodes with purplish nodules the size of lentils on the cheek. Large numbers of budding cells were cultured. Histologic examination showed epithelioid and plasma cells and many giant cells, within which were the fungi. The patient died approximately two years after the onset of the disease, of cerebrospinal involvement. Yeastlike fungi were cultured from the spinal fluid, brain, sputum, urine, palatal fistula and thigh.

The organism cultured from the thigh and that cultured from the spinal fluid were identical, whereas that obtained from the brain showed a difference in color. The cultures sent by Zach showed that when cultured on agar these fungi developed into elongated cells, as described in a previous paper,[9] and hence that they were not typical of C. histolyticus. They resembled more the organism causing the

6. Versé, M.: Ueber einen Fall von allgemeiner Blastomykose beim Menschen, Verhandl. d. deutsch. path. Gesellsch. **17**:275, 1914.

7. McGehee, J. L., and Michelson, I. D.: Torula Infection in Man, Surg., Gynec. & Obst. **42**:803, 1926.

8. Urbach, E., and Zach, F.: Generalized Torulosis, Arch. f. Dermat. u. Syph. **162**:401, 1930.

9. Moore, M.: A Study of Endomyces Capsulatus Rewbridge, Dodge and Ayers: A Causative Agent of Fatal Cerebrospinal Meningitis, Ann. Missouri Bot. Gardens **20**:471, 1933.

European type of blastomycosis of Bussey and Buschke. The organism from the sputum acquired a violaceous color when cultured on agar, which may account for the color of the lesions in the mouth and jaws. This organism differed from the others. A red yeast, isolated from the lungs and from the urine, had definitely no relationship to the others, except possibly, to a certain degree, in morphology. The fungus from the palatal fistula differed in that it was much smaller than the others.

It is possible that there were several complicating infections in the one patient, but it could not be said that the disease was a typical torulosis both of the brain and of the skin. Certainly, the microbes did not indicate the relationship. This, however, does not detract from the clinical value of this interesting case.

In 1932, at a meeting of the Atlantic Dermatological Conference (November 11), Weidman [10] reported a case of experimental torulosis in a monkey. In the discussion one of us (Dr. Mook) referred to the case described in this paper.

Three years later Wile [1] reported a case of cutaneous torulosis in a boy. In addition to unusual patches of lesions on the backs of the thighs and behind the knees, the patient had psoriasis and multiple verrucae. The peculiar lesions were hard and infiltrated. A biopsy of material from a nodule showed many giant cells and a large number of yeastlike, double-contoured budding cells. There apparently was no inflammatory reaction. It is unfortunate that a mycologic study of the parasite could not be made in this case. The illustrations accompanying Dr. Wile's paper point to an organism which may be closely related to C. histolyticus, but the response of tissue, the huge number of giant cells and the particular morphologic characteristics of the cell within the tissue indicate that a different organism may have been involved. In any case, the possibility of cutaneous torulosis is not as rare as the literature seems to indicate.

The case reported here is of interest for two reasons: 1. The lesion, which occurred on the toe, was conical and crusted and could not be differentiated clinically from an ordinary infectious granuloma. 2. Histologic examination showed a number of typical organisms of torulosis of the brain, with huge gelatinous or mucoid capsules, occurring in the skin, both in the corium and in the epidermis.

## REPORT OF CASE

*History and Course.*—The patient, a well developed man, aged 43, entered the Barnes Hospital on Jan. 27, 1932. He complained of pain in both eyes; attacks of dizziness; lack of hearing, which had been present for two months and was

10. Weidman, F. D.: Cutaneous Torulosis in a Monkey, Arch. Dermat. & Syph. **27**:720 (April) 1933.

particularly marked in the left ear; pain in the back of the neck for one and a half months, and poor sleep for one month.

The patient came to the United States at the age of 22 and worked in an iron foundry for fifteen years. During that time his hearing began to fail, and he noticed a diminution in sound effects. There was no history of any injury or buzzing in the ear. The deafness was accentuated in 1931, when the patient's older son died of tuberculosis. For five years before admission to the hospital the patient had worked in a shoe factory, and during that time he complained of neuralgia-like pains. Relatives stated that since the death of his son the patient had become reticent and was not his former self. He also complained of diminution in vision and of some blurring of objects. This gradually increased, and for a month and a half he had trouble at his work. The last month before admission to the hospital he became restless and could not sleep at night because of the pain in both eyes. The last two weeks he had attacks of dizziness while at work. He never fainted or vomited, but he was nauseated and had to lie down.

On admission there were severe headaches, chiefly in the occipital and frontal regions, which were worse at night. There was evidence of a progressive loss of vision, with occasional diplopia. The patient complained of attacks of dizziness and pain in the eyeballs and in the back of the neck; he stated that the objects in the room appeared to move away from him. He pointed to his forehead as the region of pain. His deafness gradually increased until he could barely hear shouting. He complained of ringing in the ears and had increasing vertigo. His appetite became increasingly worse, but the lack of appetite was not associated with pain, vomiting, constipation or the presence of blood in the stools. It was, however, associated with attacks of dizziness. Friends noticed that the patient's speech was slower than normal. There were no lapses of memory and no defects of speech.

In summary, the pathologic findings were: bilateral amblyopia associated with vertigo, diplopia, choking of both optic disks, obtundity of disk sensations, vertical nystagmus and upper gaze; bilateral tinnitus and deafness, the impairment being greater on the left side; some slowness of speech, and a questionable Babinski sign on the right side.

Roentgen examination of the skull was made on January 28. Lateral stereoscopic films of the skull (right side of the skull to the film) and anteroposterior films (Towne position) were made. The skull was symmetrical; the sella was within normal limits. There was increased density in the parietal region, which was somewhat greater than normal, and a slight convulsional atrophy was noted throughout the calvaria. There were symmetrically placed large areas of calcification within the region of the choroid plexus in both lateral ventricles. There was a small amount of calcification in the midline, which appeared to be in the region of the pineal body. There was a slight suggestion of erosion of the dorsum sella. The film taken in the Towne position added nothing to the foregoing observations, though it corroborated the localization of an intracranial calcification noted in lateral stereoscopic films. There was no evidence of erosion or change in the petrous portion of the temporal bone on either side.

The roentgen diagnosis was increased intracranial pressure.

The patient's stool was found to contain Hymenolepis nana (rat tapeworm).

*Operation.*—On Feb. 3, 1932, surgical intervention was resorted to. Tribromethanol in amylene hydrate supplemented by local anesthesia was used, and a flap was outlined, beginning in the medial line in the forehead and going back

to a point above the ear on the right side and then down into the temporal fossa. The flap was reflected; the dura was not very tight. In the anterior region it felt soft, as though there might be a cyst beneath it. An incision was made, and the cortex was examined, but no cyst was found. The olfactory nerve was exposed, but no tumor was found. The frontal lobe was reexamined, higher up, and a tumor about the size of a pea was found in the cortex. An area about the size of a 25 cent coin was circumscribed and cut out when it was thought that sufficient depth had been allowed for the excision. A tumor, the size of a small cherry, was removed in toto. It resembled a glioma.

*Pathologic Examination of Excised Tissue.*—The tumor was examined in the laboratory of the Barnes Hospital on February 3.

Macroscopic Examination: The material consisted of a fairly well demarcated bluish-gray tumor, about 1.5 cm. in diameter and harder than the surrounding brain tissue.

Microscopic Examination: Sections stained with hematoxylin and eosin showed a lesion with a definite outline of demarcation from the surrounding brain tissue.

Fig. 1.—Lesion of cutaneous torulosis on the second toe.

The lesion was composed of a loose tissue having in it many round bodies, which varied in size. Some were slightly larger than the lymphocytes, and others had a diameter equal to that of a moderate-sized blood vessel. Around each body was a clear area devoid of tissue. Each round body (organism) had a large granular central area, surrounded by an area which was refractile with hematoxylin and eosin but stained deep orange with phosphotungstic acid. In the surrounding tissue were many round cells, plasma cells and fibrinosis. No polymorphonuclear leukocytes were present. There was a slight increase in glial nuclei in the adjacent brain substance.

A diagnosis of torulosis of the brain was made.

*Subsequent Pathologic Observations.*—Examination of the spinal fluid on Feb. 26, 1932, showed C. histolyticus.

One of us (Dr. Mook) noticed that on the plantar surface of the second toe of the right foot there was a definitely elevated tumor-like mass, 0.6 cm. in diameter, conical, crusted and a little scaly (fig. 1). The lesion was infiltrated; it was considered to be due to a local infection, probably staphylococcic. Dr. Mook diagnosed it as an infectious granuloma and advised actual cautery.

The tumor was excised at the Barnes Hospital on Feb. 29, 1932. Grossly it was seen to consist of small pieces of reddish-gray friable tissue. Microscopic

sections showed small pieces of squamous epithelium and corium. In the corium there was an infiltration of lymphocytes, plasma cells and leukocytes. Throughout the tissue were many round bodies, varying in size. Around each there was a clear zone with a large granular area in the center. Some of these bodies were found in the epithelium, but the greatest number occurred in the epithelial connective tissue (figs. 2, 3 and 4).

A diagnosis of torulosis of the skin was made.

*Discharge and Subsequent History.*—The patient was discharged under protest on Feb. 29, 1932.

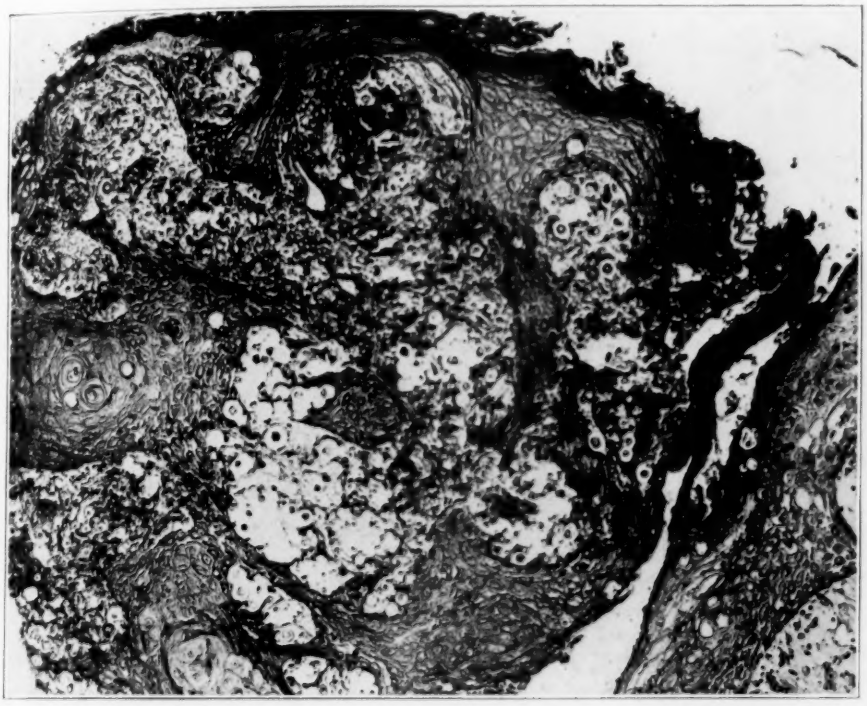

Fig. 2.—Low power magnification of a section of skin stained with hematoxylin and eosin, showing many organisms in the corium.

The patient was readmitted on March 4, 1932. During the interim, while he was at home he had a great deal of headache in the frontal region; the pain was most pronounced in the suborbital region and was practically constant. These headaches had become so severe that the patient had difficulty in sleeping at night. There had been no noticeable visual disturbances, convulsions, seizures or progressive weakness during this period. The patient's appetite had been poor, and marked constipation had been present. He returned to the hospital because of the intractability of the aforementioned symptoms.

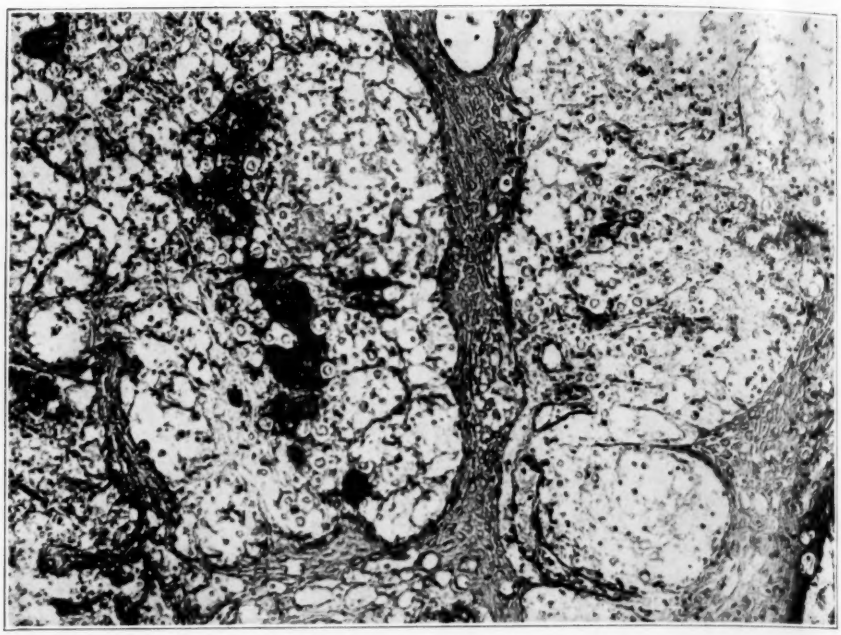

Fig. 3.—Low power magnification of a section of skin stained with hematoxylin and eosin, showing a fibrosed condition and many encapsulated fungi in the epidermis and corium.

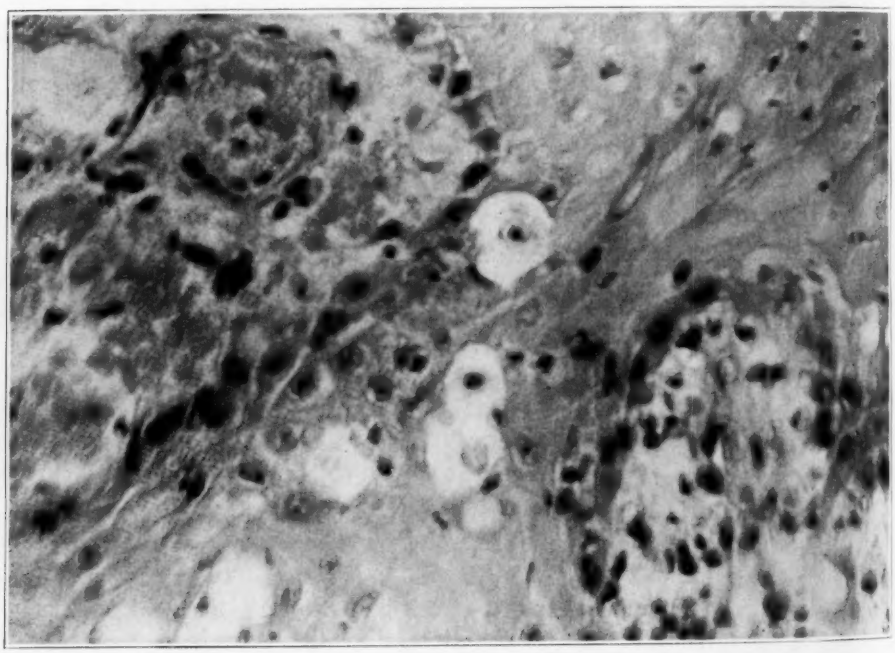

Fig. 4.—High power magnification of a section stained with hematoxylin and eosin, showing C. histolyticus.

The positive findings were: subtemporal decompression on the right side, with absence of marked bulging; bilateral choked disk; facial weakness on the left side; bilateral deafness; an Oppenheim sign on the left side, and a Romberg sign.

On April 10 it was noted that the patient was rapidly failing. He had several pressure spots on the back. He was becoming more and more stuporous. Choking of the disks had increased tremendously.

By May 1 decubitus ulceration was worse and was present over each trochanter. The patient talked at times; he took food fairly well. The fundi showed marked choking, with great profusion of hemorrhages and exudate.

The patient died on June 13.

*Final Note.*—Dr. Sachs summarized the case as follows: The disease ran a course similar to that reported in the literature on torulosis. The unusual features in the case were: (1) the presence of cutaneous lesions due to C. histolyticus; (2) the presence of a rat tapeworm which caused some confusion in the diagnosis in early stages of the disease.

*Autopsy.*—The following conditions were found at autopsy: (1) generalized torulosis involving the skin, brain, lungs and kidneys; (2) otitis media on the right side with involvement of the mastoid cells; (3) defect in the frontal lobe from an old craniotomy; (4) subtemporal decompression; (5) herniation of the right orbital lobe; (6) scaling of the myocardium; (7) clubbing of the fingers; (8) moderate arteriosclerosis, and (9) chronic pneumonia and bronchiectasis.

## COMMENT

The organism isolated from the spinal fluid, brain and lesion on the toe were identical and morphologically and physiologically conformed to the characteristics of C. histolyticus. Several months after isolation and culture the fungus showed no mycelium or endosporulation. The carbohydrate reactions were still characteristic. Unfortunately, the cultures dried and could not be subcultured. We feel certain, however, that the microbe was C. histolyticus.

There is much discussion as to the correctness of the terms Torula and Cryptococcus in reference to the causative agent of torulosis. Owing to usage, the term Torula has rapidly supplanted the correct term Cryptococcus. This change would not be at all incongruous were it not that the genus Torula has a distinct and entirely different appearance from that shown by the organism of the disease torulosis. The original description of the genus Torula given by Persoon[11] reads: "effusa, acaulis, filis articulatis indeterminate effusis mucidis." Lindau,[12] in Rabenhorst's Kryptogamen Flora, gave the following more complete description of this genus:

sterile hyphae lacking or mould-like, spread out, branched, septate, hyaline or darkly colored. Conidiophores either completely lacking or developed as short branches. Conidia developed either through disarticulation of the complete

11. Persoon, D. C. H.: Synopsis methodica fungorum Göttingen, 1801, p. 693.

12. Lindau, G., in Rabenhorst: Kryptogamen Flora, Pilze VIII, 1904-1907: Fungi imperfecti, Leipzig, Eduard Kummer, 1907, p. 567.

filaments as gemmae or by sprouting on the mycelium and then multiplying as budding colonies, or by growing out in long chains at the base of the tips of mycelial branches. The conidia are then comprised of attached chains, which finally break apart in single cells or short, jointed segments, mostly black, brown, olive-green or gray; spherical, elongated, ovoid or spindle-shaped; smooth or rough to warty.

The following material taken from Dodge [13] discusses exactly the existing knowledge with reference to Cryptococcus.

The aforementioned description differs completely from that given originally for the genus Cryptococcus by Kützing [14] in his "Algarum aquae dulcis germanicarum Decas III" and then in "Linnaea" [15] as follows: "Globuli mucosi hyalini non colorati microscopici in stratum indeterminatum mucosum facile secedens sine ordine aggregati." He gave as his type species Cryptococcus mollis Kützing occurring "on moist and dirty windows."

The description is accompanied by an illustration of a specimen dried to a small square of glass. The fungus consists of hyaline, thin-walled spores. There is also present, obviously as a contaminant, a small amount of a slender mycelium with small, slightly colored, thick-walled spores, probably a saprophytic species of Absidia, which agrees with the specimen of Kützing's Leptomitus plumula issued in Decas I, no. 9. The exact habitat is not given, but, judging from the fact that Kützing referred to this species as found in conjunction with his L. plumula, it seems probable that the organism was found on dirty windows, always in the shadow from the building opposite, in the courtyard of an inn in Herbsleben, a hamlet of Gotha, Germany. Kützing, still laboring under the misconception that these groups are polymorphic, thought that his species of Cryptococcus was close to the primordial slime of the philosophers and that various higher forms might develop from it, for he mentioned that on a well lighted window Oscillatoria developed and gradually replaced the Cryptococcus colony. Since, in general, the species of Oscillatoria require a considerable amount of organic nitrogen for their active development, it seems quite likely that the habitat where he found his species of Cryptococcus, namely, in water of condensation on windows of farmers' living rooms and inn yards, might easily have been contaminated by dirt from the stables. This supposition is also strengthened by the presence in the type collections of tiny, brown particles of dirt, which under the microscope appear to be bits of dung. Hence, it is quite possible that from the first the generic name Cryptococcus has been associated with an imperfect yeast

13. Dodge, C. W.: Medical Mycology, St. Louis, C. V. Mosby Company, 1935.

14. Kützing: Algarum aquae dulcis germanicarum Decas III, no. 28, 1833; cited by Dodge.[13]

15. Kützing: Linnaea, 1833, vol. 8, p. 365.

from the intestinal tract, if not with some more active parasite of the domestic animals.

Kützing mentioned in his publication a number of other species of Cryptococcus, but in 1849 he again discussed the genus in his "Species Algarum" [16] and still expressed the opinion that the typical members are spherical, shiny and found in moist habitats.

In the following years the generic name fell into disuse, owing, in part, to lack of interest on the part of mycologists and to the misconceptions of Persoon's Torula and Mycoderma by Desmazières, Turpin and Hansen.

In 1901 Vuillemin revived the name Cryptococcus for the asporogenous, nonfermenting yeasts from animal substrata, and since then the name has been used by the medical profession. The genus may be described as follows: The cells are spherical, ovoid or ellipsoid, occurring singly or held in more or less irregular groups by the secretion of thick, gelified capsules, especially in old age. The organism does not form ascospores, mycelium or pseudomycelium. On liquid mediums, the pellicle is thick, formed by the coalescence of slimy, floating islets when present, and the sediment is usually slimy. There is no fermentation with carbohydrates, and acidity is rare. Liquefaction of gelatin is slow when present.

Stoddard and Cutler,[3] under the guidance of Wolbach, first brought the pathologic picture to general attention, but they did not attempt to isolate the organism from material obtained from their patients and based their experimental work on species of this genus isolated more than a decade previously from lesions in the lung of a horse. They assumed that an organism which produced a pathologic condition in experimental animals similar to that found in man must necessarily be the same species as that with which they had produced the disease in man. The work of Freeman [4] indicates that there are probably three, or perhaps more, organisms which produce the same general pathologic condition, differing in minor respects. However, until there is more careful correlation of pathology and cultural characters, the nomenclature of this group must be unstable. Up to the present, only about half the pathologists who reported cases have described cultures of the organism isolated, and in not a single case has it been adequately described, while the only adequate description is not accompanied by a case history or a description of the pathologic changes.

It can be easily seen from these original descriptions that Cryptococcus is the correct generic name and should in all cases be used in preference to Torula.

16. Kützing: Species Algarum, Leipzig, p. 145, 1849.

SUMMARY

A case of fatal torulosis of the brain with a distinct and definite involvement of the skin is reported.

The known, published cases of cutaneous torulosis are reviewed with comments.

The cutaneous lesion is described as resembling an infectious granuloma.

The original descriptions of Torula and Cryptococcus are discussed, and it may be concluded that C. histolyticus is the correct term to be used for the causative agent of torulosis. This does not imply that C. histolyticus is the only species, however, for there may be other species of Cryptococcus that cause this same condition.

This report represents the third definitely known case of cutaneous torulosis, bringing the total of such cases possibly to five.

# PUSTULAR PSORIASIS

## ITS RELATION TO ACRODERMATITIS CONTINUA VEL PERSTANS

FRANCIS A. ELLIS, M.D.

BALTIMORE

In 1888 Radcliffe Crocker [1] first described acrodermatitis repens. Two years later Hallopeau [2] reported a case of chronic suppuration of the fingers of a patient who had Raynaud's disease. The pustules were not confined to the digits affected by the vasomotor disturbances but were present on other fingers and on the mucous membrane of the mouth. The lesions were superficial; they involuted rapidly and dried within twenty-four hours. Two years later Hallopeau reported another case, in a patient who had had a benign form of psoriasis since childhood. The pustular eruption appeared on the left thumb after an injury caused by a blow of a hammer. Within two years the eruption spread and involved the other digits and the palm. Staphylococci were present in the lesions.

More recently the literature has been reviewed and added to by Ingram,[3] Audry,[4] Dore,[5] Barber and Eyre,[6] Barber,[7] Bloom [8] and others. Bloom reported four cases of so-called pustular psoriasis of the palms and soles in patients who did not have psoriasis on any other part of the body.

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Presented before the Baltimore-Washington Dermatological Society, Feb. 21, 1935.

1. Crocker, H. Radcliffe: Diseases of the Skin, London, H. K. Lewis & Co., Ltd., 1888, p. 128.

2. Hallopeau, M. H.: Sur une asphyxie locale des extrémités avec polydactylite suppurative chronique et poussées éphémères de dermatite pustuleuse disséminée et symétrique, Bull. Soc. franç. de dermat. et syph. **1**:39 (June) 1890.

3. Ingram, John T.: Acrodermatitis Perstans and Its Relation to Psoriasis, Brit. J. Dermat. **42**:489 (Nov.) 1930.

4. Audry, C.: Les phlycténoses récidivantes des extrémités (acrodermatites continues de Hallopeau), Ann. de dermat. et syph. **2**:913, 1901.

5. Dore, S. E.: Note on Cases of a Chronic Mild Localized Type of Acrodermatitis Perstans, Brit. J. Dermat. **40**:12, 1928.

6. Barber, H. W., and Eyre, J. W. H.: Acrodermatitis Continua (Hallopeau) Vel Dermatitis Repens (Crocker), Brit. J. Dermat. **39**:485, 1927.

7. Barber, H. W.: Acrodermatitis Continua Vel Perstans (Dermatitis Repens) and Psoriasis Pustulosa, Brit. J. Dermat. **42**:500, 1930.

8. Bloom, David: Pustular Psoriasis of the Palms and Soles: Report of Four Cases, Arch. Dermat. & Syph. **32**:90 (July) 1935.

A controversy arose as to the exact status of this disease and its relation, if any, to psoriasis, or more specifically to pustular psoriasis. Various investigators have in the presentation of cases attributed the condition to staphylococcic infection, psoriasis and neurogenic disturbance, and some have considered it a disease sui generis. Barber, with his extensive experience, has attempted in his contribution to the literature to give a comprehensive picture of pustular psoriasis and acrodermatitis continua (Hallopeau) vel repens (Crocker), but occasionally a patient is seen whose condition defies definite differentiation and seems to bridge the gap between these two diseases. Such a case is presented here.

The superficial pustules, the granulating, distorted ends of the fingers and the fixed, useless hand are shown in detail. Note that the phlyctenules extend into the psoriasis on the flexor surface of the forearm. A biopsy specimen was taken from the area marked *A*.

## REPORT OF A CASE

C. B., a white man aged 40, was first seen at the St. Agnes Hospital, in the service of Drs. Bloodgood and Cohn, in December 1933. Ten years before, while the patient was living on a farm in Canada, his left hand was severely frost bitten. Soon afterward pustules developed about the nails of the left hand, and these had persisted up to the time of the patient's admission to the hospital. At times the condition tended to improve during the summer, but the tendency had been for it to become continually worse. Large plaques of psoriasis developed on the scalp, chest, abdomen and upper extremities eighteen months before the patient's admission to the hospital. The left arm was extensively involved, and the phlyctenules on the hand and wrist extended up into the psoriatic tissue. The entire left hand and wrist were covered with many superficial pustules or a pale pink inelastic skin. Several nails of the left hand were missing, leaving rounded granulating

stumps at the ends of the fingers, while other nails were small, distorted, rough, brittle and thickened. The hand and fingers were fixed and useless. On physical examination no other abnormality was disclosed.

The Wassermann reaction of the blood and the reaction to 0.1 cc. of a 1:50 solution of trichophytin given intradermally were negative. The blood was essentially normal. Culture of material from unruptured phlyctenules repeatedly yielded a pure growth of staphylococci. Mycelia or yeast were not seen on direct examination or in cultures. Roentgenograms of the left hand showed atrophy of the bones due to disuse, but no evidence of periostitis was discernible.

The numerous biopsies of specimens from the tips of the fingers, which were made in order to rule out the possibility of the presence of a malignant process,

*Salient Features of Acrodermatitis, Pustular Psoriasis and Condition in the Case Reported**

| Acrodermatitis Continua | Pustular Psoriasis | Condition in Case Reported |
|---|---|---|
| 1. Unilateral infective post-traumatic lesions situated at ends of digits | 1. Usually bilateral lesions; seldom any history of injury | 1. Unilateral lesions; left hand and fingers affected after severe frost-bite |
| 2. Lack of symmetry even if bilateral | 2. Striking symmetry | 2. Unilateral lesions |
| 3. Involvement of nails, early and prominent, with loss of nails | 3. No involvement of nails, or psoriasis of nails | 3. Involvement of nails from start, with loss of some nails |
| 4. Frequent involvement of mucous membranes | 4. No involvement of mucous membranes | 4. No involvement of mucous membranes |
| 5. More or less circular elementary lesions; rapidly enlarging subcorneal abscesses, which strip up the horny layer as they extend peripherally | 5. Elementary lesion: reddened areas, in which form intra-epidermic pustules, the size of a pinhead, some of which may become confluent, the majority drying up in situ to form minute brownish "scabs" | 5. Elementary lesions: often annular; rapidly formed subcorneal abscesses which raised the horny layer as they extended |
| 6. Staphylococci abundant in cultures | 6. Sterile cultures | 6. Abundant pure growth of staphylococci on repeated culture (mediums inimical to ordinary strains were not used) |

* The first two columns are adapted from the table given by Barber.[7]

showed only chronic inflammatory tissue with many fibroblasts. A specimen for biopsy was taken from the flexor surface of the left forearm, where several pustules were located in a plaque of psoriasis. Under low magnification dense collections of leukocytes were seen just under the stratum corneum, with a slight amount of fluid in some of the small pustules. Only rarely did the infiltrate penetrate into the rete. The even, regular acanthosis was striking; the pegs usually approached within four or five rows of cells of the horny layer. These pegs contained a moderate number of leukocytes, but about the tips of the interpapillary pegs the infiltrate was denser. An unusual feature was the number of leukocytes and fibroblasts in the middle and deeper parts of the corium. Many dilated hyperplastic capillaries also were present in these parts of the cutis. Under high magnification little intracellular and intercellular edema was seen in the epidermis, even in the areas adjacent to the subcorneal pustules. The leuko-

cytes seemed to migrate through fissures in the rete (tunneling of Civatte[9]). The infiltrate in the cutis was composed predominantly of polymorphonuclear leukocytes, with many early fibroblasts about the dilated capillaries. The collagen was fragmented or split into long narrow ribbons, and the elastic tissue was completely destroyed.

The salient histologic features were the large Muro-Sabouraud abscesses, the lack of edema in the rete, marked regular acanthosis and the extensive early fibrosis of the cutis.

## COMMENT

Barber[7] presented a table showing the points in the differential diagnosis betwen acrodermatitis continua and pustular psoriasis, which is given in abridged form here (table), with the addition of a third column showing the salient features in the case reported. The condition in this case differed from acrodermatitis continua in only one essential feature, that lesions were not present on the mucous membranes. It should be noted that involvement of the mucous membranes is often lacking in cases of acrodermatitis continua. Bloom expressed the belief that pustular psoriasis and acrodermatitis continua are identical.

## SUMMARY AND CONCLUSION

Although Barber seemingly gave a convincing, clearcut differential diagnosis between acrodermatitis repens vel continua and pustular psoriasis, occasionally a condition is seen which defies differentiation. A case of psoriasis is reported in which the condition was complicated by a unilateral superficial pustular eruption after severe frost-bite of the hand. The eruption had the features which Barber considered characteristic of acrodermatitis continua. From the literature and the case reported one must conclude either that acrodermatitis continua is a form of pustular psoriasis or that the patient had had acrodermatitis continua for ten years and psoriasis developed later.

104 West Madison Street.

9. Civatte, A.: Psoriasis and Seborrhoeic Eczema: Pathological Anatomy and Diagnostic Histology of the Two Dermatoses, Brit. J. Dermat. **36**:461, 1924.

# FAMILIAL XANTHOMA

## REPORT ON THREE OF FIVE SIBLINGS WITH XANTHOMA TUBEROSUM MULTIPLEX

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Seventeen reports on familial xanthoma, comprising a total of fifty-two cases, have been published.[1]

The condition is transmitted as a recessive characteristic and may be inherited through either parent. Six times it has been reported as occurring in two siblings and no other relatives,[2] and twice it has been noted in three siblings and no other relatives.[3] Two authors have recorded cases in which four siblings were affected.[4] In one family the condition was found in two brothers and their two first cousins,[5] and in another family, in three brothers and their two first cousins.[6]

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From the Department of Dermatology and Syphilology, Cleveland City Hospital, Harold N. Cole, Director.

1. (*a*) Mackenzie: Tr. Path. Soc. London **33**:370, 1882. (*b*) Startin: ibid. **33**:373, 1882. (*c*) Ehrmann: Beitr. z. klin. Chir. **4**:341, 1889. (*d*) Lehzen and Knauss: Virchows Arch. f. path. Anat. **116**:84, 1889. (*e*) Thiberge: Ann. de dermat. et syph. **5**:318, 1894. (*f*) Leskien: Inaug. Dissert., Leipzig, 1903. (*g*) Gwynne: Rep. Soc. Study Dis. Child. **5**:318, 1905. (*h*) Arning: Arch. f. Dermat. u. Syph. **105**:290, 1910. (*i*) Linser: München. med. Wchnschr. **67**:85, 1920. (*j*) Beeson, B. B., and Albrecht, P. G.: Contribution to Study of Xanthoma Tuberosum, Arch. Dermat. & Syph. **8**:695 (Nov.) 1923. (*k*) Pautrier and Levy: Presse méd. **31**:728, 1923. (*l*) Gordon and Feldman: J. Michigan M. Soc. **23**:231, 1924. (*m*) Gilbert, Chabrol and Bénard: Bull. et mém. Soc. méd. d. hôp. de Paris **22**:1067, 1926. (*n*) Wile, U. J., and Duemling, W. W.: Familial Xanthoma, Arch. Dermat. & Syph. **21**:642 (April) 1930. (*o*) Hufschmitt and Nessman: Ann. de dermat. et syph. **1**:462, 1930. (*p*) Van den Bergh, A. A. H., and Jordans, G. H. W., in Contributions to the Medical Sciences in Honor of Dr. Emanuel Libman, New York, International Press, 1932, vol. 3, p. 1151. (*q*) Lane, C. Guy, and Goodman, Joseph, Jr.: Xanthoma Tuberosum, Arch. Dermat. & Syph. **32**:377 (Sept.) 1935.

2. Startin.[1b] Ehrmann.[1c] Lehzen and Knauss.[1d] Thiberge.[1e] Gwynne.[1g] Wile and Duemling.[1n]

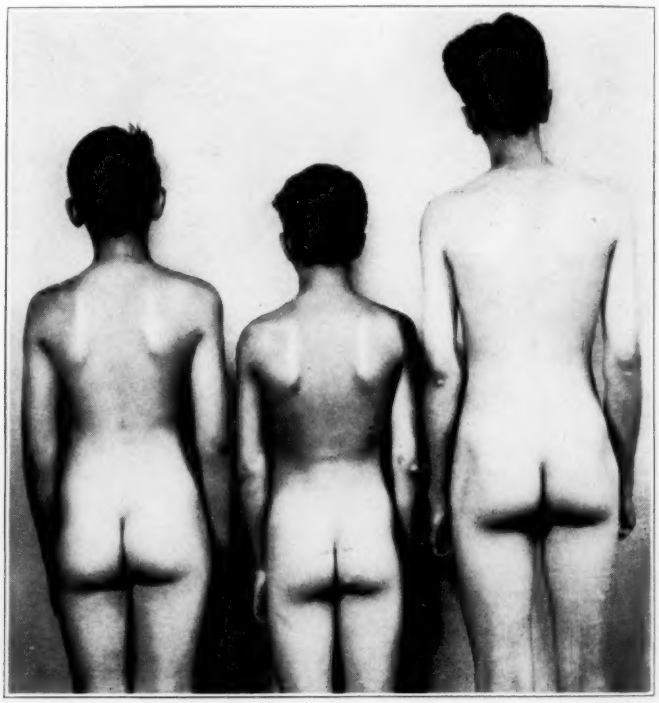
3. Leskien.[1f] Pautrier and Levy.[1k]

4. Linser.[1i] Hufschmitt and Nessman.[1o]

5. Van den Bergh and Jordans.[1p]

6. Gilbert, Chabrol and Bénard.[1m]

There are three reports of xanthoma in two generations; the first was that of a mother and four children,[7] the second that of a mother and three sons [8] and the third that of a mother and one son.[9] Most interesting is the first mention in the literature of familial xanthoma which includes members of the first and the third generation, but not the second generation, namely, the report of the condition in two brothers and a sister and paternal grandfather. To these is added the following report.



Photograph of the patients, showing xanthoma. Note the lesions on the elbows of two of the patients and the lesions on the buttocks of two.

### REPORT OF CASE

Three apparently healthy white boys, brothers, 12, 13 and 15 years old, respectively, were brought to the clinic by their father to have some yellowish tumor masses removed. There were two other siblings—a boy and a girl—but they were not similarly affected. No other members on either side of the family were known to have had any tumor masses resembling these, and there was a com-

7. Arning.[1h]
8. Beeson and Albrecht.[1j]
9. Gordon and Feldman.[1l]

pletely negative history of tumor throughout three generations. The past histories of the boys were uneventful. Each had had measles, mumps and pertussis, but no other illnesses and no operations. With the exception of the cutaneous findings nothing abnormal or remarkable was found on complete physical, ophthalmoscopic and fluoroscopic examination.

Studies of the blood lipids [10] showed the data given in the table, allowing for normal persons 0.18 Gm. of cholesterol per hundred cubic centimeters of blood plasma, 0.2 Gm. of lecithin and 0.8 Gm. of total fat.

In these patients urinalyses and Wassermann tests of the blood gave negative results. The blood picture was normal in the 13 year old boy, but showed a basophil count of 6 per cent in the 12 year old boy and of 20 per cent in the 15 year old boy. In each case the urea nitrogen, blood sugar during fasting, sugar

*Cholesterol, Lecithin and Total Lipoid Values per Hundred Cubic Centimeters of Blood Plasma for the Three Patients*

| Patient | Age, Years | Date | Cholesterol, Gm. | Lecithin, Gm. | Total Fat, Gm. |
|---|---|---|---|---|---|
| D. R. | 12 | June 25 | 0.438 | | |
| | | June 28 | 0.443 | | |
| | | July 7 | 0.483 | | |
| | | July 12 | 0.428 | | |
| | | July 17 | 0.460 | 0.670 | 2.08 |
| R. R. | 13 | July 12 | 0.390 | | |
| | | July 17 | 0.467 | 0.642 | 2.76 |
| E. R. | 15 | July 10 | 0.480 | | |
| | | July 12 | 0.425 | | |
| | | July 17 | 0.409 | 0.589 | 2.42 |

tolerance reaction and basal metabolic rate were within normal limits. Biopsies in all three instances showed a typical histopathologic picture of xanthoma.

## CONCLUSIONS

There are added to the literature on familial xanthomatosis reports of the cases of three of five siblings with xanthoma tuberosum multiplex.

The family history on both sides was negative for xanthoma.

The blood pictures of two of the patients showed the percentage of basophils to be markedly increased.

In all three patients the values for cholesterol, lecithin and total lipoid were abnormally increased.

10. The determinations were made by Dr. Ramon F. Hanzal, Department of Biochemistry, Western Reserve University.

# AMYLOIDOSIS CUTIS LICHENOIDES

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AND
A. W. FRESHMAN, M.D.
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It is only recently that a report of a case of localized cutaneous amyloidosis appeared in the American dermatologic literature, although this condition has been familiar to European writers for about fifteen years. In Germany and Italy, Juliusberg, Koenigstein, Gutmann, Sannicandro and others have reported more than thirty cases, and it is probable that it occurs with equal frequency in this country but has escaped general recognition.

Virchow named the substance occurring in this condition amyloid because of the starchlike reaction which usually resulted when the lesions were treated with aqueous solution of iodine and dilute sulfuric acid. Bonetus [1] in the seventeenth century referred to the homogeneous, glassy substance as wooden induration. From Rokitansky came the designation lardaceous degeneration.

Kekulé [1] (1858) and Friedreich [2] classified amyloid substance with the group of albuminoids. This classification Schmidt confirmed in the following year. A step forward resulted from the work of Krawkow, who regarded the substance as a combination of albumin and chondroitin and sulfuric acid. Although this chemical conception has found its way to modern textbooks, Lubarsch [3] stated that there are unclear and disputed points that need elucidation. Eppinger,[4] working with masses of pure amyloid, failed to recover sulfur, phosphorus or carbohydrate. The substance differs in many respects from hyalin, with which it is confused. The latter is a variable substance

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Read before the regular monthly conference of the staff at the sanatorium of the Jewish Consumptives' Relief Society, Spivak, Colo., May 8, 1935.

1. Kekulé, cited by Trambusti, A.: Patologia della cellula, in Foà, Pio: Trattato di anatomia patologica per medici e studenti, Torino, Italy, Unione tipografico editrice Torinese, 1922, p. 117.

2. Friedreich, in Ribbert, M. W. H.: Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie, Leipzig, F. C. W. Vogel, 1908, p. 115.

3. Lubarsch, O.: Die hyalinen und amyloiden Ablagerungen, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 1, p. 459.

4. Eppinger, cited by Wallace, J. E.: Diagnosis of Amyloid Disease, Lancet **1**:391 (Feb. 20) 1932.

chemically and morphologically and as to origin. It has a tendency to stain with acid aniline dyes, while amyloid reacts characteristically to methyl green and methyl violet.

It was first pointed out by Heschl [5] (1879) that methyl green is the reagent for amyloid. His observations were confirmed by Curschmann (1880), who showed that it stains amyloid an intense violet. It is known that good results can be obtained with Cornil's method, using tissue fixed with a solution of formaldehyde.

Amyloid has a marked affinity for congo red. Congo red cannot be used with tissue fixed with a solution of formaldehyde, as the color will be lost. The injection of congo red intravenously to stain amyloid was suggested by Bennhold [6] in 1923. The amount of dye recovered from the plasma is an index of the extent of lardaceous degeneration, and it has recently been shown that congo red injected subcutaneously beneath the lesions will find its way to amyloid deposits in the corium.

## REPORT OF A CASE

H. L., a writer aged 47, from the Sanatorium of the Jewish Consumptives' Relief Society, was first seen in the hospital service of Dr. A. J. Markley on Feb. 20, 1935, with a complaint of an itchy eruption on his legs. No diagnosis was made on this visit, and the patient was requested to return for further study. On his second visit he gave the following history:

The family history was irrelevant except for the fact that his father had had an itchy condition of the legs for many years. The patient went to school until 16 years of age and then became a teacher. He was in prison in Siberia for seven years and came to America in 1913. There was a past history of typhus in 1910, Vincent's angina in 1911 and influenza in 1912. The patient was of the opinion that his lungs were affected at the time of the attack of influenza. He had furunculosis from 1929 to 1932. He stated that he had not had a venereal disease. He had had no injuries or operations.

The patient dated the onset of pulmonary tuberculosis from October 1922, when he began to notice cough and expectoration, increased pulse rate and subfebrile temperature. A diagnosis of pulmonary tuberculosis was made at that time, and after a rest of one year he returned to his work. He tired easily, coughed and was very susceptible to colds. In 1932 he began to suffer from chills, night sweats and increased cough and expectoration, and at that time he came to the sanatorium of the Jewish Consumptives' Relief Society. The clinical course of the patient in the sanatorium was uneventful, and the condition was diagnosed as far advanced tuberculosis of both lungs. Roentgen diagnosis confirmed the physical findings of far advanced chronic fibrocavernous tuberculosis, with slight activity in the upper lobe of the right lung. The patient was ambulant and afebrile, and the symptoms were practically nil. Examination of the sputum gave negative results. His only complaint was frequency of urination, associated with occasional pain, and

5. Heschl, cited by Lee, A. B.: Microtomist's Vade-Mecum, Philadelphia, P. Blakiston's Son & Co., 1905, p. 197.

6. Bennhold, H.: Ueber die Ausscheidung intravenös einverleibten Kongorotes bei den verschiedensten Erkrankungen insbesondere bei Amyloidose, Deutsches Arch. f. klin. Med. **142**:32, 1923.

for this prostatic massage was given. After residence of six and one-half months he left the sanatorium, with the tuberculous lesions quiescent and with the prognosis favorable. After he was discharged the patient stayed in New York for one and one-half years, at the end of which time he felt worse. He therefore returned to Spivak, Colo., on Sept. 6, 1934. Examination revealed no change in his general condition and that of his chest. There was a moderate dry cough but no expectoration, and the disturbances of urination had subsided completely. The patient was ambulant and afebrile. He stated that he had suffered from itching all over the body all his life. During the past five years the itching had

Fig. 1.—The anterior surface of the legs on which the majority of lesions are located.

become worse and had concentrated especially on both legs. Two years previously a papular eruption developed on both lower extremities, which started with the ankles and progressed toward the hips.

*Laboratory Observations.*—The sputum could not be examined because there was no expectoration. The sedimentation rate was 3 mm. the first hour and 9.5 mm. the second hour. The vital capacity was 121 per cent. The red blood cell count was 4,700,000; the white blood cell count was 7,900; the hemoglobin content was 80 per cent, and the color index was 0.8. The differential count was: segmented forms 60 per cent, lymphocytes 33 per cent, monocytes 1 per cent, staff

...ils 4 per cent, and eosinophils 2 per cent. Examination of the urine gave nega... results. The reaction to the Wassermann test was negative.

*Dermatologic Examination* (O. S. P.).—There were no cutaneous abnormalities ...ther than a papular eruption of the legs of about two years' duration. This eruption (fig. 1) was symmetrically distributed, and the lesions were more abundant on the flexor than on the extensor surfaces. On the ankles the lesions were closer together, larger and more numerous than elsewhere. It was here that the condi-

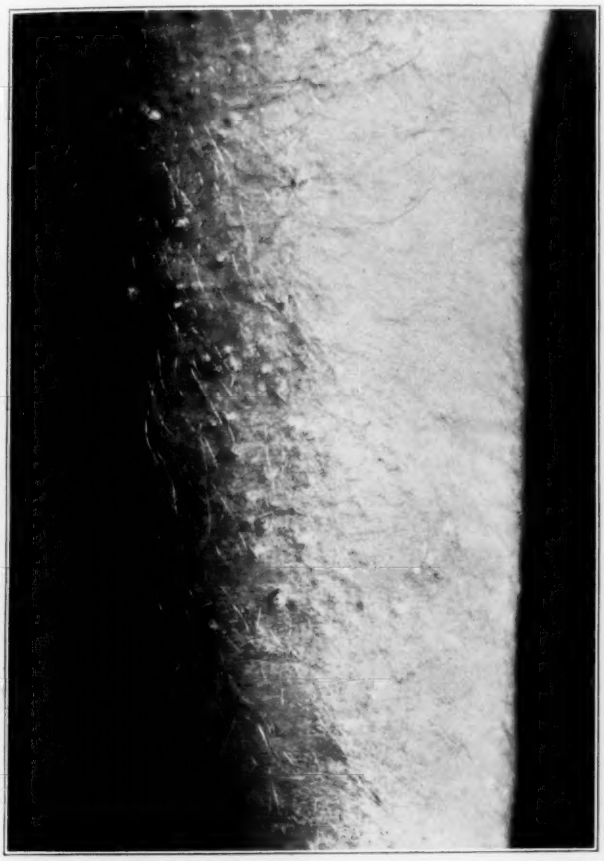

Fig. 2.—The flexor surface of the legs showing papular lesions—some verrucous, some crusted and occasionally one with a hair protruding through the top.

tion first appeared, gradually spreading upward, the most recent lesions appearing as papules the size of a pinhead on the knees. The eruption was entirely papular, although some lesions, appearing to be vesicular or slightly translucent, were firm and hard. They were faintly yellow, some being slightly pink or reddish brown. They varied from the size of a pinhead to that of a matchhead and were conical or hemispherical. Many were capped (fig. 2) with a verrucous scale, and on others such scales had been removed or replaced by bloody crusts as the result of scratching. A few lesions formed in the follicular orifices, and through each of these a hair protruded.

*Diagnosis.*—The conditions considered in the diagnosis were: lichen planus, lichen simplex chronica, verruca vulgaris, Darier's disease, pityriasis rubra pilaris and molluscum contagiosum.

Study of the material taken for biopsy, however, at once excluded all these possibilities. The sections presented unusual features, evidently the result of an infiltrative process. Search of the literature for analogous conditions disclosed reports [7] of two cases of localized cutaneous amyloidosis in which the clinical and microscopic observations were practically identical with those in the present case. After this by special staining methods, the diagnosis of the condition was made. The subcutaneous injection of congo red beneath several typical lesions, as recently suggested by Nomland,[8] resulted in their being stained a bright red, which persisted for several weeks. Bennhold's intravenous test with congo red revealed a 29 per cent decrease after one hour, indicating the absence of any sys-

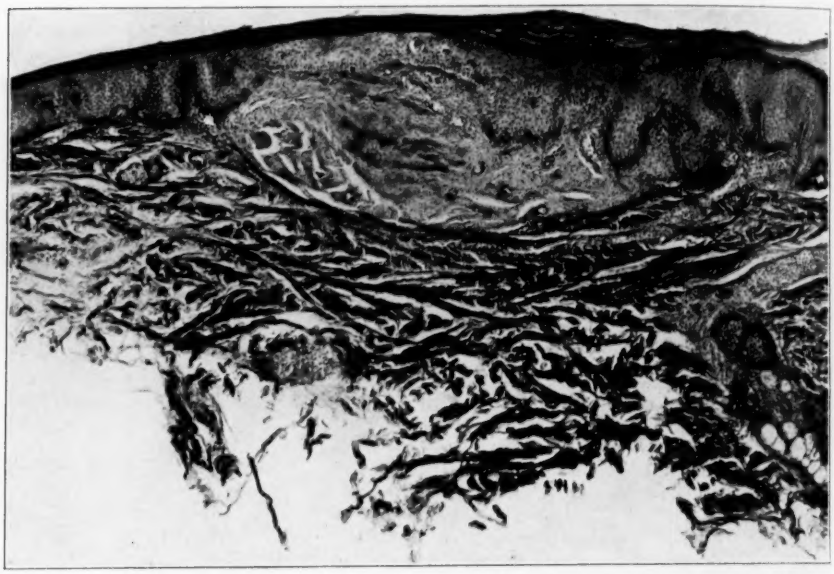

Fig. 3.—A photomicrograph (× 40) of a frozen section fixed in a solution of formaldehyde and stained with hematoxylin and eosin. It shows atrophy of the epidermis and disappearance of the rete cones. Hyperkeratosis is present. The amyloid nodule is seen beneath the epidermis in the papillary bodies.

temic amyloidosis, since it has been demonstrated that amyloid deposited in the skin does not absorb the dye when it is administered intravenously.

*Histopathologic Picture of the Cutaneous Lesions* (A. W. F.).—Frozen sections stained with hematoxylin and eosin (fig. 3) revealed large nodular masses consisting of broad wavy bundles of homogeneous-staining material, poor in cellular ele-

7. Winer, L. H.: Local Amyloidosis of the Skin, Arch. Dermat. & Syph. **23**:866 (May) 1931. Ormsby, O. S.: Diseases of the Skin, ed. 4, Philadelphia, Lea & Febiger, 1934, p. 672.

8. Nomland, R.: Localized (Lichen) Amyloidosis of the Skin, Arch. Dermat. & Syph. **33**:85 (Jan.) 1936.

nodules, beneath the epidermis. The rete cones had disappeared. There was a thinning of the horny layer overlying the lesion. Other areas showed well developed papillary bodies, with smaller nodules separated by well developed rete cones. The homogeneous material was deposited about the thin-walled capillaries in the papillary bodies. In the upper layer of the cutis was moderate round cell infiltration.

Sections stained with orcein (Grübler's) showed absence of elastic fibers in and about the homogeneous areas.

Unfixed frozen sections stained with methyl violet showed the nodules in the corium to be deep red; the surrounding tissue was blue.

Sections (fig. 4) treated with aqueous solution of iodine U. S. P. showed the nodules to be a mahogany color, which changed to light blue, or grayish blue, when washed in dilute sulfuric acid.

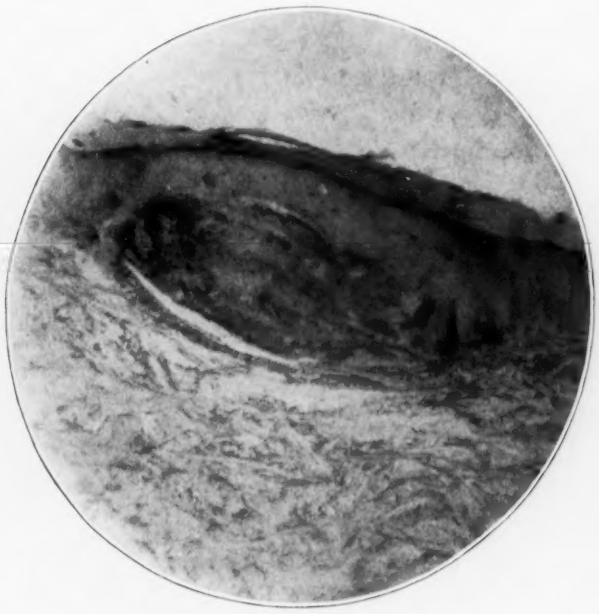

Fig. 4.—Photomicrograph of an unfixed frozen section stained with iodine. It demonstrates the amyloid nodule in the papillary bodies, with thinning of the epidermis and disappearance of the rete cones.

A subcutaneous injection of 1 cc. of a 1 per cent solution of congo red was made beneath two lesions, and microscopic examination of unfixed material from these lesions forty-eight hours later revealed the amyloid substance in the corium to be stained light red, with the surrounding tissue colorless.

## COMMENT

This case demonstrates the difficulty of proving by routine staining methods a clinical diagnosis of amyloidosis cutis, and it emphasizes the need for further investigation into the nature of amyloid, particularly in regard to its reactions to stains. It demonstrates, further, the need

for persistent and repeated attempts to prove the presence of amyloid, even after one or two apparent failures. The results reported in the literature and in the present case prove that an examination in a suspected case of amyloidosis cutis is not complete unless numerous staining methods are tried and their results are reported.

SUMMARY

A case of amyloidosis cutis lichenoides has been reported in a man who had pulmonary tuberculosis for many years.

A section of fixed tissue stained by routine methods failed to demonstrate conclusively the presence of amyloid, but tissue treated by the methods outlined in this paper gave definite and positive evidence of its presence.

A subcutaneous injection of congo red gave a positive vital stain to the lesions, even after the disappearance of the rosy-hued congo red stain in the surrounding skin.

227 Sixteenth Street.

# SPHACELODERMA

## REPORT OF A CASE OF UNUSUAL PHAGEDENIC ULCERATION OF THE SKIN, SUBCUTANEOUS TISSUE AND MUSCLE OF THE CHEST WALL

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Gangrene of the skin [1] (sphaceloderma) may be single or multiple, acute or chronic, local or general (some types are peculiar to certain areas),[2] symmetrical or asymmetrical, small or extensive. Gangrene may be a sequela to one of a variety of conditions, or it may be spontaneous.[3]

Crocker stated: "Single gangrenous patches, often of large size, are met with in infants and young children, both spontaneously and as a result of infectious fevers. They start as a vesicle, pustule or bulla."

Gangrenous lesions caused by the ingestion of iodides were reported by R. Parker in a girl aged 10 years and by Audrey in a woman aged 47. Wende reported a fatal case, in which bullous lesions developed in a chronic ulcer on the leg. These subsequently became gangrenous. Streptococci, staphylococci, diplococci and bacilli were found in the bullae and in the gangrenous lesions during life and in the internal organs at autopsy. A case reported by Waelsch also proved fatal. After the use of a morphine syringe gangrenous patches and abscesses developed in a man aged 38. He died in three weeks. A bacillus was

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From the surgical division of Dr. Edgar Burke and from the dermatological department of Dr. Wilbert Sachs, Jersey City Medical Center.

1. Crocker, H. R.: Diseases of the Skin, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1903, p. 527. Stelwagon, H. W., and Gaskill, H. K.: Diseases of the Skin, ed. 9, Philadelphia, W. B. Saunders Company, 1921, p. 427. Ormsby, O. S.: A Practical Treatise on Diseases of the Skin, ed. 4, Philadelphia, Lea & Febiger, 1934, p. 356.

2. Hilbert, R.: Spontaneous Gangrene of the Eyelids, Vrtljschr. f. Dermat. **11**:117, 1884. Corbus, B. C.: Erosive and Gangrenous Balanitis, J. A. M. A. **60**:1769 (June 7) 1913. Gaté, J., and Rousset, J.: Gangrene of the External Genitalia Caused by Spironema Vincentii, Ann. de dermat. et syph. **10**:151 (Feb.) 1929. Palmer, H. E.: Gangrenous Vulvitis or Noma Pudendi, Florida M. A. J. **16**:31 (July) 1929. Chevallier, P. D.; Lévy-Bruhl, M., and Moricard, R.: Idiophagedena of the Vulva (Geometric Phagedena of Brocq and Simon), Bull. Soc. franç. de dermat. et syph. **38**:212 (Feb.) 1931. Peacock, S. C.: Dry Gangrene of the Face, with Mummification and Separation *en Bloc* of the Nose and Adjacent Tissues, Am. J. Dis. Child. **45**:815 (April) 1933.

3. Smith, L. D.: Spontaneous Gangrene of the Extremities, Am. J. Dis. Child. **44**:151 (July) 1932.

found that did not stain by Gram's method. A fatal case was reported by Lancashire. The condition involved the lower part of the abdomen, the inguinal regions and the inner surface of the upper part of the thighs. There was chronic eczema in the involved region. Bacillus pyocyaneus and Staphylococcus aureus were found. In a case reported by Hartzell, vaccinia-like lesions, later becoming gangrenous, developed in a woman, aged 46, after an injury with a poisoned meat-hook. In the bullae and in the sloughs from the gangrenous areas many bacilli and staphylococci (Staph. aureus) were found. The case reported by Hallopeau started with acne-like papules, which quickly exfoliated, leaving an ulcer. Gangrenous ulcers are usually small, but in the cases of most severe involvement they may slough and suppurate and extend over a large area. The condition occurs most frequently on the face, but it may affect mucous membranes and the mouth and even the chest and arms. All these cases were cited by Crocker.

Cases of extensive ulceration caused by amebas were reported by Engman and Heithaus, by Engman and Meleny and by Crawford.[4] In the first two cases the condition developed after incision of an amebic abscess and after a surgical intervention on the intestinal tract, respectively. In the case reported by Crawford it was associated with amebic colitis, but no surgical procedure was done. The condition starts as a very painful tender abscess, which ruptures and discharges a thick pus. The edges become undermined, and about them there is a red areola. Amebas may be found in the wound and in the feces.

Dermatitis gangraenosum infantum,[5] or *ecthyma télébrant,* is a gangrenous eruption which follows varicella and other pustular eruptions and occasionally occurs spontaneously. Stelwagon stated that it occurs in patients under the age of 3 years, appearing most commonly in the first year. The condition starts as pinhead-sized papulopustular multiple lesions, which become crusted and beneath which ulceration begins. The process may extend peripherally, or lesions near each other may become confluent, and large areas may be involved. When the condition arises spontaneously it usually begins about the buttocks. Extensive lesions are usually associated with marked constitutional involvement (temperature 104 F., vomiting, etc.) and pulmonary and cardiac complications, and as a rule the termination is fatal. Mild lesions may have few concomitant symptoms.

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4. Engman, M. F., and Heithaus, A. S.: Amebiasis Cutis, J. Cutan. Dis. **37**:715 (Nov.) 1919. Engman, Martin F., Jr., and Meleney, Henry E.: Amebiasis Cutis (Endamoeba Histolytica): Report of Two Cases, Arch. Dermat. & Syph. **24**:1 (July) 1931. Crawford, Stanley: Amebiasis Cutis: Report of a Case, ibid. **28**:363 (Sept.) 1933.

5. Pasachoff, Harry D., and Sobel, Nathan: Dermatitis Gangraenosa Infantum in the Course of Scarlet Fever, Arch. Dermat. & Syph. **26**:428 (Sept.) 1932.

Cases of postoperative progressive gangrene were reported by Baker and Terry, Horsley, Cullen, Lynn, Freeman, Christopher, Bremer and Meleney, Ballin and Morse.[6] The ulceration usually follows operation, starting around a stitch. The affected area becomes painful, swollen and purple and quickly ulcerates. This condition rapidly spreads. The subcutaneous fat, fascia and skin are involved. The temperature is only moderately elevated, even when the wound is extensive. The etiologic factor in these cases is thought to be a symbiotic synergistic infection with two types of bacteria. A similar condition was described by Horsley,[7] which did not follow operation. However, the patient had urinary disturbances; his condition was more toxic (vomiting, high pulse rate, some fever) than that noted in the other cases, and the wound spread more rapidly.

Pyoderma gangraenosum, as described by Brunsting, Goeckerman and O'Leary, by McCarthy and Fields and by Lane and Stroud,[8] has a definite relationship with infections of long standing elsewhere in the body. There are improvement and recurrence, with similar changes in the general condition. The primary lesion is a papule or a small nodule, which becomes encrusted and ulcerated. The lesions are multiple and vary in diameter from 1 cm. to 14 cm. Staphylococcus albus and a streptococcus were found in the lesions.

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6. Baker, W. H., and Terry, C. C.: A Case of Postoperative Progressive Gangrene of the Skin, J. A. M. A. **98**:138 (Jan. 9) 1932. Horsley, J. S.: Intussusception Due to Intestinal Lipoma in an Adult, Followed by Gangrene in the Abdominal Wall, Arch. Surg. **18**:882 (March) 1929. Cullen, T. T.: A Progressive Enlarging Ulcer of the Abdominal Wall Involving the Skin and Fat Following Drainage of an Abdominal Abscess, Apparently of Appendiceal Origin, Surg., Gynec. & Obst. **38**:579 (May) 1924. Lynn, F. S.: Postoperative Gangrenous Ulcer of the Abdominal Wall, J. A. M. A. **97**:1579 (Nov. 28) 1931. Freeman, Leonard: Progressive Gangrenous, Painful Ulceration of the Abdominal Skin and Subcutaneous Tissues Following Operation, Ann. Surg. **92**:779 (Oct.) 1930. Christopher, F.: Severe Spreading Carbuncular Infection of Chest Wall Following Rib Resection Under Local Anesthesia, S. Clin. North America **4**:795 (June) 1924. Bremer, G. E., and Meleney, Frank L.: Progressive Gangrenous Infection of the Skin and Subcutaneous Tissues Following Operation for Acute Perforative Appendicitis, Ann. Surg. **84**:438 (Sept.) 1926. Ballin, Max, and Morse, P. F.: Progressive Postoperative Gangrene of the Skin, Am. J. Surg. **11**:81 (Jan.) 1931.

7. Horsley, J. Shelton: Certain Symbiotic Bacterial Infections Producing Gangrene, with Special Reference to the Principles of Treatment, J. A. M. A. **98**:1425 (April 23) 1932.

8. Brunsting, L. A.; Goeckerman, W. H., and O'Leary, Paul A.: Pyoderma (Echthyma) Gangrenosum: Clinical and Experimental Observations in Five Cases Occurring in Adults, Arch. Dermat. & Syph. **22**:655 (Oct.) 1930. McCarthy, Lee, and Fields, Russel: Pyoderma Gangraenosum, New York State J. Med. **31**: 801 (July 1) 1931. Lane, Clinton W., and Stroud, Malone C.: Pyoderma Gangraenosum: Report of Case, Arch. Dermat. & Syph. **27**:460 (March) 1933.

Nocardiosis cutis gangraenosa was described by Guy and Helmbold.[9] The patient had a septic temperature and bloody dysentery. The lesions were multiple and very painful. With the appearance of the lesions the patient had a rise in temperature, chills, nausea, anorexia and general symptoms of an acute infection. To a great extent the condition is similar to pyoderma gangraenosum, except that the organism Nocardia was recovered from the lesions. Gangrene due to hemolytic streptococci follows a cutaneous wound, gangrene occurring several days after the injury and spreading rapidly to include the subcutaneous tissue. This has been described by Meleney, Jen, Bates and Fallon.[10]

## REPORT OF CASE

M. M., a girl aged 10 years, born in New Jersey of Polish parentage, was well, aside from having had measles and an occasional cold, up to the onset of the condition described in this paper. Her parents, brothers and sisters were living and well. There was no history of tuberculosis, diabetes or syphilis in the family.

During January 1931 she was treated at the Jersey City Medical Center in the outpatient department of the surgical division for a "furuncle" of the left axilla. This was incised and found to be purulent; several days later the lesion became necrotic and began to slough. During February 1931 a superficial fluctuating area developed left of the sternum at the level of the second and third ribs. This was incised and drained and, like the axillary lesion, in a few days became necrotic and began to slough and to extend peripherally.

The patient was admitted to the hospital on March 17, 1931. At that time there were two ulcerated areas; one covered the entire medial surface of the left arm to within 2 inches (5 cm.) of the cubital fossa and extended over the entire left axilla and the lateral surface of the chest almost down to the twelfth rib; the other extended from the left margin of the sternum up to the midaxillary line. The two ulcerated areas were separated by a 1 inch (2.5 cm.) strip of undermined skin. The base of the ulcer showed a red granulating surface with some purulent secretion, and in places the pulsations of the heart were clearly visible. The margins were undermined, and the skin and subcutaneous tissue were raised off the muscle, with purulent necrotic material between (fig. 1).

The general condition of the patient was good and continued so, with the exception that her temperature at all times ranged between 99 and 100 F. She was confined to bed, with her arm raised in a splint in the attempt to avoid a contracture between the arm and the chest wall, but later, when she was allowed to be up, her appetite and color improved. (During an epidemic of diphtheria in the ward she contracted the disease but recovered rapidly.)

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9. Guy, W. H., and Helmbold, T. R.: Nocardiosis Cutis Gangraenosa, Arch. Dermat. & Syph. **27**:224 (Feb.) 1933.

10. Meleney, Frank L.: Hemolytic Streptococcus Gangrene, Arch. Surg. **9**: 317 (Sept.) 1924. Jen, T. K.: Hemolytic Streptococcus Gangrene, China M. J. **43**:889 (Sept.) 1929. Bates, John T.: Subcutaneous Streptococcus Gangrene, Ann. Surg. **90**:1029 (Dec.) 1929. Fallon, John: Hemolytic Streptococcal Subcutaneous Gangrene: Report of Case, Arch. Surg. **18**:1817 (April) 1929.

*Laboratory Examination.*—All tests were repeated several times. The Wassermann reaction of the blood was negative. Blood counts showed from 15,000 to 17,000 white cells and from 3,000,000 to 4,000,000 red cells. The hemoglobin content was from 40 to 50 per cent, and the color index was 0.6. The sputum was normal at all times, and the throat was normal except when the patient had diphtheria. There were no gonococci. The stools contained no amebas. No reaction was obtained when a guinea-pig was inoculated. A smear of material from the wound contained no tubercle bacilli, amebas or fusiform bacilli. Staphylococci but no fungi could be cultured. Biopsy disclosed granulation tissue, which was of no special type.

Roentgenograms showed nothing abnormal, except that on Jan. 25, 1932, there was a trophic condition of the bones of the shoulder, the humerus and the upper part of the radius and ulna. There was a proliferative process of the inner aspect of the shaft of the humerus from the surgical neck downward about 2 inches (5 cm.). On June 6, 1932, the chest showed a generalized infiltration throughout

Fig. 1.—Photograph of the patient at about the time of admission to the hospital.

the entire right lung and the upper lobe of the left lung, which was suggestive of infection with the tubercle bacillus. There was osteomyelitis, involving the upper half of the left humerus.

*Therapy.*—Hydrogen peroxide, physiologic solution of sodium chloride, hypertonic solutions of sodium chloride up to 10 per cent and bacteriophage were used as wet dressings at various times, each being given a fair trial period. The edges of the lesion were painted with silver nitrate at first; later pure lactic acid was used. From May 1931 to February 1932, she was exposed to radiation from a carbon arc lamp three times a week for twenty minutes at each sitting. At the same time she was exposed to the sun's rays daily. She was given eight injections of antimony and potassium tartrate to rule out amebiasis and leishmaniasis. She received a high caloric diet, viosterol, beef hemoglobin and liver extract in an alcoholic menstruum, iron and ammonium citrate, potassium iodide, solution of potassium arsenite and solution of iron and ammonium acetate. One blood transfusion of 300 cc. was given in the attempt to improve her resistance.

*Subsequent History.*—The patient's general condition remained good throughout. The osteomyelitis of the left humerus became progressively worse, and on Dec. 15, 1932, the area was operated on, and the necrotic granulations were curetted. She responded well to this, and the condition gradually improved.

The wound remained more or less stationary during 1933 and 1934, with the occasional appearance of healthy granulations at several points. In December 1934 she went home for two weeks for the Christmas holidays. She returned to the hospital, and since that time the wound has slowly but progressively improved, until at the time of writing the entire area has undergone epidermizations.

The treatment was continued throughout the entire time as at the start; the use of each of the various remedies was repeated many times. At no time before or during the healing was any special treatment used.

Fig. 2.—Photograph of the patient after the lesions had healed, showing the extent of the scar formation.

All the laboratory examinations were repeated too often to tabulate. She received three blood transfusions: 300 cc. on Aug. 1, 1932, and, Nov. 30, 1932, and 350 cc. on Dec. 16, 1932.

*Condition at the Time of This Report.*—The girl is 14½ years old, is well nourished and weighs 109 pounds (99 Kg.). The osteomyelitis is completely healed. She has no subjective symptoms and, other than some impairment in motion, experiences no discomfort. There remains only a little motion in the left arm owing to adhesions to the chest wall. The left forearm has about 50 per cent motion, and there is full use of the left hand.

The left breast is gone. The right breast is present, but there are only the nipple and a small portion of the tissue of the breast about it. The outer portion of the breast is wanting. On lateral view, there is a marked concavity of the left side of the chest, causing considerable deformity of the thorax. The previously ulcerated areas are entirely healed. The scar extends across the whole wall of

the chest, the upper margin reaching to the top of the sternum in the center and on the sides and extending over the shoulders; the lower border reaches to the bottom of the chest wall. The scar extends down over the upper third of the right arm; on the left side it extends around the back, in the upper part to within 2 inches (5 cm.) of the spine and in the lower part to the lower pole of the scapula. Almost the entire left arm is covered by the scar tissue. The edges of the scar are irregular, in places white, smooth and at the same level with the surrounding skin and in places raised, well defined and red. The surface is in part smooth except for a few cordlike red bands traversing it in different directions. In the region of the left shoulder and left arm the surface is very irregular, with large masses protruding. There are large fibrous bands connecting the left side of the chest wall and the upper half of the left arm. In part the scar is smooth, stretched, thin and glossy; in part it is wrinkled, dull and not unlike cigaret paper in appearance. Over the lateral surface of the left arm is a deep scar where the incision was made because of the osteomyelitis (fig. 2).

## NOTES ON PRESENTATION AND DISCUSSION OF THE CASE

This patient was presented by Dr. David Satenstein with a diagnosis of phagedenic ulcer at the October 1931 meeting of the Manhattan Dermatological Society.[11] Later in the same month the patient was presented by me for diagnosis at the Bronx Dermatological Society.[12] In January 1934 the patient was again presented by Dr. Satenstein and me at the Manhattan Dermatological Society.[13]

The discussions were directed chiefly toward therapy; no diagnosis was made, but the following were suggested: tuberculosis, syphilis, severe scrofuloderma and *ecthyma télébrant*.

Similar cases had been observed by several of the men; in none was a diagnosis made, and none of them appear to have been reported.

Dr. Isadore Rosen stated: "I have seen two similar cases during the past few years, in which all the pathologic and bacteriologic studies failed to reveal a definite etiologic factor."

Dr. N. Sobel said: "I was fortunate enough to see a case similar to this in Jadassohn's Clinic about two years ago. The lesions were smaller, and the duration was over one year. Repeated examinations, carefully made, gave absolutely negative results, and no diagnosis could be made."

Dr. Ludwig Oulmann commented: "I saw a patient with a similar condition, who had fistulas and a rolling up of the epidermis, denuding the arm from the axilla to the wrist. The patient was tuberculous."

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11. Satenstein, D.: Phagedenic Ulcer, Arch. Dermat. & Syph. **25**:390 (Feb.) 1932.

12. Sachs, W.: A Case for Diagnosis, Arch. Dermat. & Syph. **25**:399 (Feb.) 1932.

13. Satenstein, D., and Sachs, W.: Phagedenic Ulcer of Unknown Origin, Arch. Dermat. & Syph. **30**:156 (July) 1934.

Morrow,[14] in discussing a case of dermatitis gangraenosa chronica presented at a meeting of the San Francisco Dermatological Society, mentioned several cases of a condition which corresponded closely to the one reported here.

Y. Tsutsui [15] reported the case of a man who had a large chronic progressive gangrenous lesion involving the entire anterior wall of the chest, the axillae and the upper portion of the abdomen.

### DIFFERENTIAL DIAGNOSIS

The history, clinical picture and course and laboratory findings rule out extensive ulcerations caused by tuberculosis, syphilis, actinomycosis, blastomycosis, sporotrichosis and coccidioidal granuloma.

Never was there any history of ingestion of drugs, especially iodides, before admission to the hospital. The clinical appearance and the pathologic findings were against the diagnosis of the condition as an eruption caused by drugs. The wound healed, even though the patient was taking iodides.

The cases reported by Wende, Waelsch and Lancashire were fatal; in all there was a preexisting condition; the progress was rapid, and organisms were recovered. The case reported by Hartzell followed an injury; the lesions were multiple, and organisms were found. The acne-like multiple lesions, location, duration and course are unlike those in the case reported by Hallopeau.

Amoebiasis cutis is easily ruled out because in this disease there is either an operation or a condition harboring amebas or amebas are found in the stool. Besides, antimony and potassium tartrate was given to this patient as a therapeutic test, with no results.

Dermatitis gangraenosum infantum is rarely extensive, localized and arising spontaneously. It occurs before the age of 3 years, usually first about the buttock, when not preceded by a pustular eruption, and when extensive it is accompanied by marked constitutional involvement and pulmonary and cardiac complications and is nearly always fatal. None of these features fit in with the condition in this case.

The condition in this case does not fit in with those of postoperative progressive gangrene and gangrene due to hemolytic streptococci because there was no operative intervention, the primary lesion was painful and ulcerated and extended extremely slowly and similar organisms were not found. In the case reported by Horsley there

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14. Morrow, Howard, in discussion on Templeton, H. J.: Dermatitis Gangraenosa Chronica, Arch. Dermat. & Syph. **26**:361 (Aug.) 1932.

15. Tsutsui, Y.: Ein Fall von seltener, chronischer progressiver Hautgangrän, Arch. f. Dermat. u. Syph. **85**:219, 1907.

were toxic manifestations and constitutional disturbance (urinary), and the wound spread rapidly.

Pyoderma gangraenosum is also associated with constitutional symptoms; the lesions are multiple, and the primary lesion is a papule or a small nodule. The patients are toxic at times. The largest lesions are small compared with the one in the present case. Besides, similar organisms were not found.

The condition in this case is not like those described as nocardiosis cutis gangraenosa for the same reasons given in regard to pyoderma gangraenosum. Nocardia was not found.

## COMMENT

From the foregoing data it can easily be seen that the condition in the case reported does not fit in with any of the types of gangrene described. It belongs to an undiagnosed or unclassified type of gangrene mentioned by Crocker in his textbook and by Rosen, Sobel and Oulmann in a discussion of this case and is similar to a type cases of which have been presented at meetings of various dermatologic societies. In order to summarize the many different types of gangrene, the following classification is suggested:

I. Caused by obstruction of circulation
   - (*a*) Embolism and thrombosis within vessels
   - (*b*) Changes in wall produced by
     1. Acute arteritis—bacterial, syphilitic
     2. Degeneration—senile gangrene, decubitus
     3. Physical agents—heat, cold, radiation
     4. Chemical and medicinal agents—caustics, acids, alkalis, ergot, and iodides
   - (*c*) Pressure on vessels from without owing to inflammation, effusion, extravasation of blood, scars and tumors

II. Associated with acute inflammatory disease
   - (*a*) Typhoid, malaria, pneumonia, scarlet fever and erythema multiforme

III. Associated with chronic systemic disease
   - (*a*) Leprosy, tuberculosis, syphilis and diabetes
   - (*b*) Debilitating disease—cardiac, pulmonary, hepatic, renal
   - (*c*) Gastro-intestinal diseases (especially of the colon) diseases of the blood and neoplasms

IV. Associated with nervous disorders
   - (*a*) Raynaud's disease, hysterical gangrene and syringomyelia (Morvan's disease)

V. Caused by micro-organisms

(*a*) Noma, dermatitis gangraenosa infantum, gas gangrene, gangrene due to hemolytic streptococci, gangrene from symbiotic bacterial infections, postoperative progressive gangrene, tropical phagedena (?) pyoderma gangraenosum, nocardiosis cutis gangraenosa, traumatic gangrene, actinomycosis, blastomycosis, sporotrichosis, granuloma coccidioides

VI. Unclassified

SUMMARY

A case of extensive ulceration, of spontaneous origin, in a girl aged 10 years, showing no subjective or constitutional symptoms at any time, is reported. A diagnosis has not been made, and the condition is not like that in any case which has been reported. All laboratory investigation proved of little value, and the condition resisted all types of treatment. It started without any known cause and terminated after four years without apparent cause.

All the laboratory work was done in the laboratory of the Jersey City Medical Center.

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# THE FIELD OF DERMATOLOGY

PRESIDENT'S ADDRESS

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Skin diseases occur on the surface of the body, where every one can see them, and few recognize them. Because of the first fact and regardless of the second, most physicians are inclined to attempt to treat them. The result is that the dermatologist is inclined to think that the field that he should regard as his is a free-for-all domain; and in a sense it is. There is never a time when one group or another is not undertaking to appropriate some part of it. Every one comes forward with new suggestions, usually therapeutic, for what he thinks is eczema, psoriasis, lichen planus, acne or what-have-you.

Now it is an internist, with a leaning toward intestinal disorders, who tells dermatologists how to treat cutaneous diseases by feeding the patient, or not feeding him, or by feeding him certain things. Give more sugar or give less, cut down the amount of protein or increase it, have him live on vegetables or some kind of hay. It is gout or rheumatism that is causing the trouble, or acidosis or alkalosis. Have him walk 3 miles a day in the open air, even if his feet do hurt from ringworm. Give him salicylates, arsenic, mercury, cod liver oil, vitamin D, alkalis, acids, spinach or liver; discontinue cow's milk and give him goat's milk. Starve him or stuff him. Wash out his stomach or fill him with drugs. One's style is not cramped much when one is treating hypothetic causes of diseases which one does not know by methods which one likes to use.

Again, it is the allergists who think that they are the persons to treat skin diseases, even if they cannot recognize them, because they alone know how to make patch tests, scratch tests and intradermal tests, overlooking the fact that Jadassohn, a dermatologist, initiated the whole subject by introducing the patch test in 1895, that he and his pupil Bruno Bloch, another dermatologist, developed its precise application, and that they and other dermatologists following them not only have shown its value in an important group of dermatoses but have had perhaps a larger part than any one else in indicating the importance of allergy in general pathology. The allergists busy themselves in making patch tests with innumerable substances for lichen planus, lupus,

Read at the Annual Meeting of the Chicago Dermatological Society, Jan. 15, 1936.

psoriasis, scabies, chickenpox and syphilitic eruptions and finding causes that do not exist for these and other diseases that they do not know and on the basis of their findings treating the diseases by methods that do no good. One of the difficulties is, as I once heard a puzzled physician explain, that the skin tests show that the patient is sensitized to coca cola while sarsaparilla produces his hives. The allergists have a large field because their domain is the unknown, at least the unknown to them in dermatology. Seriously, I believe that it is the experience of dermatologists that they are likely to get no help from the allergists, even when they try to work in the closest cooperation. Their technic with cutaneous diseases does not give the satisfactory findings that the dermatologists get from their own technic, and their interpretations of the results are not borne out in the dermatologists' experience. It is like trying to be useful in finding something with the appearance of which one is unfamiliar in a strange room in the dark.

The roentgenologists and the radiologists would appropriate a large part of dermatology because they are so expert in handling the necessary machinery, overlooking the fact that the dermatologists have been expert enough to do practically all the original work in developing the therapeutic possibilities and uses of the roentgen rays and radium, that their work with skin diseases pointed the way to the therapeutic application of these agents for diseases of other structures and that they even did a great deal of the original work in the treatment of these diseases of the deeper structures.

This situation reminds one of the story of the haughty father who, when a young man was seeking his daughter's hand in marriage, asked him with lofty superiority whether he could support her in the luxury to which she was accustomed, to which the young man replied, with a little irritation, for which I confess I think some justification may be found, that he ought to be able to because he had got her accustomed to it.

Or a surgeon arises who goes about preaching that all patients with cancer of the skin should come to him, for he is the person who knows how to cut out a cancer, and he continues happily in his career of cutting out lupus, chancres and gummas with the complacent idea that he is eradicating malignant growths at their beginning and so, like the boy scout, is doing his daily bit in the campaign for the control of cancer. He depends for his diagnosis on the laboratorian, both of them forgetting for the time how uncertain histologic diagnosis is without full clinical knowledge. Conservative pathologists realize the pitfalls in differentiating between epithelioma and other sorts of chronic ulcers of the skin and likewise in differentiating sarcoma from other chronic hyperplastic lesions of connective tissue, and all dermatologists can tes-

tify to the mistakes that are made in diagnosing these conditions. Indeed, I have often felt that it would be a safe rule for a surgeon to have a dermatologist pass judgment on all lesions in the skin before operation. If the surgeons think, as I have no doubt they do, that the rule should work both ways, I am willing to have a surgeon pass judgment on the treatment of the same sort of lesions by a dermatologist before this is undertaken. I am vain enough to believe that the dermatologists would be more useful to the surgeons than the surgeons would be to the dermatologists. But I am willing to accept the advantages that the dermatologists would gain through the satisfaction, perhaps not altogether altruistic, of teaching them something about the skin. I hesitate at my temerity in offering this criticism of the surgeons, for I have an impression that they are touchy about their prerogatives, and I am likely to invite a counter-blast. I would not minimize their great capacity for usefulness. I am simply pointing out that even the surgeons are not at their best in some borderline fields. One could choose, I think, no better illustration of the dangers of spreading out one's field too thinly.

And now the psychoanalysts would treat diseases of the skin by laying open with the most painstaking dissections those primitive biologic urges that we dermatologists, who are still tainted by victorian modesty, approach with reluctance because we are so unsophisticated as to feel that there are at least a few details of personal life that the individual might conceivably rather keep to himself and, indeed, might often be better off for not having emphasized in his attention before he has become a middle-aged crustacean. The psychoanalysts have one advantage: It takes so long for them to study a case that in the period of years that they require for examination the cutaneous disorders have the opportunity to demonstrate the fortunate tendency that they often show to get well of themselves.

We also have on us the endocrinologists and the physical therapeutists, with all their machinery, and the laboratorian, who is a bacteriologist in his first phase and an immunologist in his terminal phase, who apply their particular methods in any cases that they happen to bag in the terra incognita of dermatology.

Oh, this thing that is called life is a vexatious, trying experience to the dermatologists!

There is one consolation about it all: We are used to it, and with patience one can get used to almost anything. All physicians have to get used to it more or less. There is nothing new in the situation. Dear old Bishop Berkeley had a panacea in his tar water. Then Perkins with his tractors, mesmerism and hypnotism, galvanism, homeopathy and an unending list of treatments based on theories of disease have

filed past. This state of affairs has been, after superstition, perhaps, the greatest stumbling block to medical progress. As Huxley said, "Science commits suicide when it adopts a creed." Old Bonomo answered all of them when he commented, apropos of his discovery of the itch mite: "From this discovery [that the itch mite causes scabies] it may be no difficult matter to give a more rational account of the itch than authors have hitherto delivered us. It being very probable that this contagious disease owes its origin neither to the Melancholy Humour of Galen nor the corrosive acid of Sylvius, nor the particular ferment of van Helmont, nor the irritating salts in the serum or lymph of the moderns, but is no other than the continued biting of these animalcules in the skin."

It is a sad fact, but true, that the itch is still treated on the assumption that it is due to "the Melancholy Humour of Galen." It is a melancholy humor all right. And that is where the poacher gets into trouble. It does not do any good for the amateur or the professional psychoanalyst to bring out, after laying open his secret, that the young man's heart is bowed down by fear because of his one fateful indiscretion. The laborious discovery of his secret will do no good, and it misleads the psychoanalyst. One cannot reason with the itch; one cannot cast it out from a mind diseased, and one cannot even cure it with arsphenamine, under the impression, gathered from the history, that the patient really has syphilis.

That is the difficulty about cutaneous diseases: One has to know what they are. Dermatologists have to bear a great deal of joking about their elaborate and complicated nosology and nomenclature. If our ridiculers knew how to diagnose a few cutaneous diseases it would not seem so amusing to identify them and have names for them. The ludicrous thing is that scientific men do not realize that differential diagnosis is as necessary for diseases of the skin as for diseases of the kidneys or liver, for example.

The inexorable necessity for making diagnoses is the difficulty that undoes all the groups who would treat cutaneous diseases (or any other diseases) by method rather than on the basis of diagnosis. This necessity for making diagnoses is the difficulty—and the salvation—of dermatology. Treatment by method does not work, and so one sort of enthusiasts follows another. But the work of dermatologists, barring treatment of a certain fairly fixed percentage of mystical patients who must have something different, preferably something exotic, continues in the hands of those who know the itch when they see it, can tell lupus erythematosus from rouge, do not treat impetigo with serums and do not treat lupus vulgaris, gumma and epithelioma all in the same way.

Treating by method rather than on the basis of diagnosis does not work well anywhere in medicine. The endeavor of medicine and all science for the last hundred and fifty years at least has been to secure factual knowledge, in contrast to hypothetic knowledge: to differentiate typhoid from typhus, chickenpox from smallpox and appendicitis from inflammation of the bowels before it is too late, and to recognize that an organism and not some form of struma is the essential cause of tuberculosis and that boils are due to pus organisms and not to "bad blood." It is this sort of scientific effort that brings about progress in medicine. If dermatology, because of the accessibility of its field, has gone further than most other specialties in differentiating pathologic processes, it is a scientific feat and a credit. And, too, this has made dermatology a great deal more useful. It is not our fault that the finer detailed knowledge in the field is highly technical and requires an elaborate nomenclature; that is equally true of chemistry or botany, for example. There is only one thing that must be done by those who wish to cultivate the subject—that is to learn it. Of course, no group has any right to claim a natural monopoly of any field. The only claim that we can have on dermatology must be that we have better knowledge of it than others. If we cannot keep that position we do not deserve to control it and in time we will not.

But diseases of the skin are our field; it is regrettable that dermatology is so difficult to learn, but its very difficulty keeps the one-methodists from taking much of it for long.

In addition to the clinical complexity of dermatology, another fact marks it as a definite field: That is the fact that so large a proportion of cutaneous diseases not only are confined to the skin but are of local origin or are not involved in any disturbance of the general health, so far as can be determined, and one must know dermatology to recognize these. I am not saying, and I do not believe, that there are not many diseases of the skin that are produced or influenced by internal factors, but I am ready to maintain that physicians, including dermatologists, are inclined to overemphasize the importance of internal causes, that the more that is learned of diseases of suspected internal origin the more important local causes appear to be and that the internal treatment of cutaneous disease of unknown origin usually gets one nowhere.

Engman has given a fine phrase, "the skin, the mirror of the body." He had in mind that the skin shows practically all the pathologic processes that occur in the body and that in the skin they are exposed to direct view. From another standpoint one might with equal truth speak of the skin as the mask of the body. In a small group of systemic diseases the skin characteristically reveals internal disease, in a few others it does so in rare cases, but as a rule, far from being a gossip

about what goes on inside, it is a discreet protector of the body's secrets. Think of the vast multitude of conditions in which the skin is faithful to its function as a cover, concealing the distempers within: nephritis, diabetes, cardiac and vascular disorders, tuberculosis, dyspepsia, carcinoma and even syphilis and nervous and mental diseases. It is not pleasant to think what a leprous-looking lot human beings would be if the skin really reflected the conditions inside the body. There is no more difficult part of dermatology than the interpretation of the general health from the condition of the skin and the interpretation of the condition of the skin from the general health.

And that is another reason that usually renders useless the treatment of undiagnosed cutaneous disease by the employment of measures which are empirically directed to defects in the general health which are surmised to exist or which, if existent, are actually irrelevant to the cutaneous disorder which the patient may have. One can make the "high liver" and the plethoric person unhappy by treating them for their gout or gouty diathesis as the cause of their cutaneous disorders; one can cut off their liquor and put them on buttermilk, and still their psoriasis, pruritus, ringworm or eczema goes on. One can feed iron to pasty-looking girls (who may or may not have anemia) until the iron market is affected. One can give constipated or listless patients yeast until one opens up unexpectedly a great outlet for that formerly modest and, in its place, useful commodity (as I believe has actually been done), and their acne, muddy complexion or eczema persists. This method of treatment does not work. One has to know the diseases that one is treating in the practice of dermatology. That is why dermatologists were born. And that, I doubt not, is why they will continue to exist for some time.

Is there any reason for joy to dermatologists in that thought? Even if we have a field of usefulness, is it a worth-while field; does it give its devotees in a reasonable degree the three sorts of reward—material, emotional and intellectual—that men have a right to expect in intellectual vocations?

I have had occasion recently to summarize this topic in another address, and I shall briefly refer to a few points here. The skin offers the great advantage that in it normal and pathologic tissues and functions are open to observation in their natural relation. Fortunately, also, as Auspitz long ago pointed out, "The skin's pathological features . . . correspond entirely to the diseases of organs in general." It is in this sense that the skin is a mirror of the body. This fact has long been appreciated in medicine and has been specifically pointed out innumerable times.

The importance of the skin in general medicine is shown in many important illustrations. The present concepts of contagion, immunity and allergy had their beginning in the study of manifestations in the skin. Modern bacteriology and the modern methods of staining used in bacteriology and histology were first applied in studies of the skin. Manifestations of the effects of vitamins on the skin have been important in the development of the present knowledge of these substances. The one definite cause of cancer that is known, besides heredity, has been demonstrated in the studies of irritation of the skin. These are only a few of the many illustrations of the great fruitfulness of the study of the skin.

Dermatology has a field. It has its borderlines, which are not at all points distinct and may merge into other fields. Along these there are likely to be friction, as this also occurs on the ill defined borders between nations. But its great area is well defined, and we possess it not by right of possession but by right of discovery and useful occupation. As long as we hold it by that right it will remain ours. We need have no particular anxiety about the inroads of the highlanders who come down and raid our borders. "The good old rule, the simple plan, that he shall take who has the power and he shall keep who can" is sound ethics when it is applied to the rights of knowledge. As long as dermatologists justify the possession of their field by superior usefulness and scientific achievement they can hold it. And no one would wish that they hold it longer.

# *BEJEL:* NONVENEREAL SYPHILIS

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Various aspects of *bejel*, the form of syphilis found among the Arabs of the valley of the middle Euphrates River, have been dealt with in previous papers from the clinic of the American Mission at Deir-ez-Zor.[1] Its nonvenereal aspect, however, has not hitherto received specific consideration, although *bejel* would seem to differ radically from conventional syphilis in this respect.

Sexual intercourse is so definitely linked with syphilis in clinical experience and scientific thought that the history of exposure to venereal disease through intercourse and the presence of genital primary lesions are recommended in textbooks to differentiate syphilis from other diseases. Syphilis without a venereal factor, though its possibility has been generally assumed and though some observers have even found it in isolated cases, has never been studied in a large population group and has remained largely hypothetic. My statement, therefore, that syphilis does exist, as *bejel,* in a purely nonvenereal form in a Bedouin population numbering 150,000, demands proof.

In this paper I have presented my grounds for the belief that syphilis in the form of *bejel* is nonvenereal. The evidence lies along four lines: 1. The Bedouin villagers are prevented from sexual promiscuity by geographic, economic and social barriers. 2. The Bedouins, large numbers of whom have been treated in the clinic, are free from venereal lesions and diseases, and such venereal lesions and diseases as have been noted have occurred in other social groups. 3. The Bedouins say that there is no promiscuity among them and attribute the widespread propagation of *bejel* in their communities to infection through innocent contact, usually in childhood. 4. Experience with the clinical course of *bejel* confirms the fact that it is a nonvenereal community disease of the Bedouins acquired during childhood.

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1. (*a*) Hudson, E. H.: Treponematosis Among the Bedouin Arabs of the Syrian Desert, U. S. Nav. M. Bull. **26**:817, 1928. (*b*) Hudson, E. H., and Young, A. L.: Medical and Surgical Practice on the Euphrates River, Am. J. Trop. Med. **11**:297, 1931. (*c*) Hudson, E. H.: Syphilis in the Euphrates Arab: A Clinical and Serological Study, Am. J. Syph. & Neurol. **16**:447, 1932; **17**:10, 1933. (*d*) Hudson, E. H., and Young, A. L.: Arab Syphilis: Further Serological Studies, J. Christian M. A. India **8**:284, 1933. (*e*) Hudson, E. H.: Juxta-Articular Nodules in Euphrates Arabs, Tr. Roy. Soc. Trop. Med. & Hyg. **28**:511, 1935.

## GEOGRAPHIC AND SOCIAL LIMITATIONS

A glance at any map of the Near East will remind one of the fertile crescent, beginning on the left with the Lebanon Mountains, running in a northeasterly curve along the south of Turkey and ending to the east in Irak. This well watered crescent enfolds a semiarid steppe known as the Syrian Desert, across which, radially to the arc, run the Middle Euphrates River and its tributary, the Khabur River. The town of Deir-ez-Zor lies near the junction of the rivers at a point about the middle of the chord subtending the crescent. The straight line length of the Euphrates River from the Turkish to the Irak border is about 250 miles (320 kilometers), and the length of the Khabur River is about 125 miles (160 kilometers).

The people who live along these two rivers are insulated from contacts with the outside world to a remarkable degree by the desert barrier, the nearest city being over 200 miles away, and most of the people still live as they have lived for milleniums. The settled population of the region numbers about 200,000, of which 195,000 are indigenous moslem Arabs.[2] This number does not include the nomad Arabs of the Syrian Desert, whose position and numbers fluctuate with the seasons and who are not included in the scope of this paper.

The Christian population of the region, numbering about 5,000, has little social significance and is not indigenous. The Christians are refugees and others whose homes are elsewhere; they are to be found only in the towns.

The indigenous Moslems who comprise the bulk of the population are divided into two distinct social groups; in one group I have placed the townsmen and in the other, the Bedouins. The townsmen number 50,000, of whom 35,000 live in Deir-ez-Zor and the remainder in four small towns on the Euphrates River, namely, Rakk, Sabkha, Meyadin and AbuKemal. Of the Bedouins, 132,000 live in small mud villages along both banks of the Euphrates River, and 13,000 live similarly along the Khabur River.

Among the Christians, the patronage of prostitution exists to a degree closely comparable to that in the West. Syphilis among them is known as *franghi* (the disease of the Frank, or westerner), and the name carries with it the connotation of venereal origin and of shame.

The name given to syphilis among the townsmen depends on its source in a given case. A townsman who has contracted syphilis through venereal contact refers to it as *franghi,* whereas childhood infection among the townspeople is given the Bedouin name of *bejel.* The infants of town families are often sent out to the Bedouin villages

2. The figures on population which are given here are all approximate but are based on an accurate census now being made by the government in Deir-ez-Zor.

to spend the first year or two of life. They may thus contract *bejel*. In other instances *bejel* may appear among the children of a town family through some contact in a village. Thus, many of the adult townspeople have had infection with *bejel* in childhood, and I have seen some cases in which it seemed likely that there had been a nonvenereal infection in childhood with *bejel* and a venereal infection in adult life with *franghi* in the same patient. In none of these cases, however, could the observations be checked scientifically.

In general, *bejel* is dying out in the towns. My associates and I see much less *bejel* in town children than we did ten years ago. Most persons in the town now receive some treatment.

The Bedouins call their type of syphilis *bejel*; it has the connotation of a childhood exanthem, and the Bedouins say they have had it without shame. They recognize that the children of their communities are the great reservoir of the acute form of the disease and that adults who have escaped the infection in childhood usually contract it from children, often their own, later in life. They state that there is no sexual promiscuity among them, and they resent any suggestion that *bejel* is the same as *franghi*, because the latter term has a venereal connotation.

Though most of the town families sprang originally from Bedouin stock, townsmen and Bedouins are now sharply demarcated by social distinctions of dress, language and mode of life. Especially are they to be distinguished in their economic status.

The townsmen comprise shopkeepers, money-lenders, landowners, middlemen, pedlers, artisans, officials and professional men of religion and law. The Bedouins in their villages along the two rivers combine cereal farming with sheep raising. They are virtually seminomads. They wage an unequal and continual struggle against floods, locusts, drought and extremes of heat and cold. The economic status of the townspeople is comparatively high, while that of the Bedouin is just above the level of starvation.

The Bedouins never live in town, seldom stay in town overnight and mingle with the townspeople only as they chaffer and bargain in the bazaars. They reach the town on donkeys or on foot, following the path along the river, and the towns of the region are so few that such journeys often consume two or three days. The villager supplies most of his own scanty needs and seldom leaves his home to dispose of his farm products. His money-lender and landowner are on hand at the harvest to take their share and bargain for the surplus. There are no fairs for trade and jollification, such as are often found among the peasantry in other countries. Few of the young Bedouin men enter the army, and none of the Bedouin children attend the town schools.

The townspeople despise the Bedouins as dirty and uncouth; the dislike is mutual, and one group can hardly refer to the other without

a curse. The Bedouin is the butt of the jokes and ill humor of the market place and is pushed off to one side in the cafés. Feeling himself at a disadvantage in the bonds of city walls and conventions, he is always glad to return to the open spaces of the river and the desert. He has not time, money or opportunity to patronize the houses of prostitution, the cinemas or the dancing entertainments of the town. Finally, he does not touch alcohol in any form. There are brothels in the towns and also much clandestine prostitution, but the women cater only to townsmen and soldiers. Bedouins are not solicited, and a Bedouin would be thrown out and probably beaten by the other patrons if he tried to enter a brothel.

Thus, the Bedouin men, owing to their own poverty and to social barriers raised against them, have practically no venereal contacts in the towns. Then how much prostitution and promiscuity is there in their own villages? Here a very severe social code operates to reduce promiscuity to a negligible quantity. The Arab, though primitive in many respects, has a highly developed sense of sexual propriety, which the closely knit clan life of the community still preserves. The Arab law on matters of sex is based on the value of the child-bearing woman as the mother of sons, who are the fighting power of the tribe. If a Bedouin man is sexually impotent his wife's family have the right to declare the marriage void and select another husband who can "raise up sons."

The wealth of a Bedouin is measured in daughters as well as in sons; in fact, from the standpoint of money, daughters are assets and sons are liabilities. In each village and tribe the value of a marriageable daughter is fixed by custom. In the region of Deir-ez-Zor the market price of a girl is from fifteen to twenty sheep, 30 bushels of wheat and a camel or 3 Turkish gold pounds. The total value would represent perhaps 22 gold pounds, or about $150. At current wages, a Bedouin working for Bedouins would require from one to two years to earn this amount in daily labor. Some of the sheiks hold their daughters for prices up to 200 gold pounds.

Though many fathers pay the marriage price for their sons' brides and many other marriages are arranged by exchange, there remain many young men who have to work years before they can afford the purchase price of a wife. Under these circumstances it is obvious that the father of daughters has a strong commercial motive reenforcing his natural repugnance to antemarital promiscuity on the part of his daughters, and he consequently guards them well.

The penalty for rape is very severe. Rape by night is punished less severely than rape by day, because in view of the distribution of the day's labor the woman is more likely to be alone during the day, whereas

at night she is supposed to be under the care of her male relatives. An unmarried woman who becomes pregnant is likely to be killed by her brothers as a matter of family honor, and if she identifies the father of the child her brothers may kill him also or exact a heavy fine. Arab law does not recognize any "age of consent" on the part of the woman. No matter how mature she may be, power of consent to her marriage is always lodged in her father or, if he is dead, in her oldest brother.

When one sees the intimate way in which the villagers live and the simplicity of their family and community life, it is difficult to imagine the possibility of much clandestine sexual promiscuity. Girls are usually married as soon as child-bearing becomes possible; large families are the rule; the woman is a responsible and important member of the family and the tribal organization, and husband and wife usually hold positions of mutual esteem. When the first wife has aged with hard work and child-bearing the man may marry another and younger woman, but this is done naturally, without derogation to the status of the first wife. The tribal organization does not tolerate an unmarried woman, and one is very rare.

To sum up these observations: Prostitution is found only in the towns; the brothel is easy of access only to townsmen. The Bedouin is not a welcome guest in the town; he lives at a great distance; he works hard the entire year; he has little money and little interest in the amusements in town; he seldom enters military service, and he does not touch alcohol. In their own close-knit communities the Bedouins do not tolerate sexual promiscuity, rape is severely punished, marriage is made by purchase, with a high cash premium on virginity, and girls are always married at an early age.

On these a priori grounds, venereal disease should be rare among the Bedouins. This conclusion is supported by clinical data.

## CLINICAL DATA

The consideration of the clinical material is divided into three parts. On the assumption that venereal disease in a given community is most likely to be found in its overt aspects among the men, I shall first discuss the syphilitic history and lesions of all the males 15 years and older admitted to the clinic in the course of 5,000 consecutive general new admissions. One may also assume that the incidence of gonorrhea in a community is a valuable check on the existence of promiscuous sexual intercourse. Therefore, in the second part of this section I shall analyze all the cases of gonorrhea in males and females observed in the clinic in the course of 7,000 consecutive general new admissions. In the third part of this section I shall indicate what I believe is the answer to the question: How then, if it is not venereal, is *bejel* propagated?

1. *Syphilitic Males.*—Proportion of Social Groups Among Patients: During the three years ending June 30, 1934, 4,355 different men, women and children were admitted to the clinic in Deir-ez-Zor. Of these, 970 (22.3 per cent) were former patients, and 3,385 (77.7 per cent) were newly admitted. Of the 3,385 new patients, 799 (23.6 per cent) were Christians, 1,202 (35.5 per cent) were townspeople and 1,384 (40.9 per cent) were Bedouins.

Proportion of the Sexes Among New Patients: Previous statistical studies have shown [3] that the new patients in the clinic were divided approximately equally between the sexes.

Proportion of Syphilitic and Nonsyphilitic Males Among 2,500 New Patients: Between June 27, 1932, and Sept. 1, 1934, 2,500 new patients were admitted to the clinic. During this period there were admitted 870 males of 15 years and older, of whom 518 (59.5 per cent) were not syphilitic and 352 (40.5 per cent) were syphilitic.

Of these 870 males, 179 were Christians, of whom 21 (11.7 per cent) were syphilitic and 158 (88.3 per cent) were not syphilitic. There were 274 townsmen, of whom 57 (20.8 per cent) were syphilitic and 217 (79.2 per cent) were not syphilitic. There were 417 Bedouins, of whom 274 (65.7 per cent) were syphilitic and 143 (34.3 per cent) were not syphilitic.

Thus, in a consecutive series of 870 men admitted to the clinic, two-tenths were Christians, with an 11.7 per cent incidence of syphilis, three-tenths were townsmen, with 20.8 per cent incidence of syphilis, and one-half were Bedouins, with a 65.7 per cent incidence of syphilis.

Observations on 660 Syphilitic Men: Between Jan. 27, 1930, and Sept. 1, 1934 (three years and seven months), exactly 5,000 new patients were admitted to the clinic. Of these, approximately 2,500 were males. The exact number of men 15 years and older was 1,763.

Of these 1,763 men 15 years and older, 660 (37.4 per cent) were syphilitic according to diagnosis and 1,103 (62.6 per cent) were not syphilitic. Of the 660 syphilitic men, 50 (7.6 per cent) were Christians, 156 (23.6 per cent) were townsmen, and 454 (68.8 per cent) were Bedouins.

Thus, though 23.6 per cent of the new patients were Christians, only 7.6 per cent of the syphilitic males were Christians. Though 35.5 per cent of the new patients were townspeople, only 23.6 per cent of the syphilitic males were townsmen. On the other hand, the Bedouins comprised 40.9 per cent of those newly admitted but furnished 68.8 per cent of the syphilitic males.

Ages of 660 Syphilitic Men: Of the 660 syphilitic men 15 years and older, 39 were between 15 and 19 years; 222, between 20 and 29 years; 203, between 30 and 39 years; 97, between 40 and 49 years; 46, between 50 and 59 years; 40, between 60 and 69 years, and 13, 70 years or older. Thus, 261 (40 per cent) were between 15 and 29 years of age, and 464 (70 per cent) were below the age of 40 years. In other words, 70 per cent of this series of syphilitic men were within the age when venereal disease is most common. One hundred and ninety-six (30 per cent) were over 40 years of age.

Anamnesis of 50 Syphilitic Christians: Of the 50 syphilitic Christian men, 1 had contracted *bejel* from the Arabs when a refugee in childhood. Eighteen gave a definite age of infection with *franghi* as follows: 3 between 15 and 19 years, 12 between 20 and 29 and 3 between 30 and 39 years. Six did not state the age of infection, 4 were uncertain, and 21 said they did not have an infection.

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3. Hudson,[1b, c] Hudson and Young.[1d]

Of the 6 who did not state the age of infection, the Kahn reaction of the blood was doubtful for 1 and positive for 5. Of the 4 who were uncertain about infection, the Kahn reaction of the blood was negative for 1 and positive for 3. Of the 21 who said they did not have syphilitic infection, the Kahn test of the blood was not carried out on 2, and the reaction was negative for 3 and positive for 16.

Anamnesis of 156 Syphilitic Townsmen: Of the 156 syphilitic townsmen, 55 (35 per cent) said they had had *bejel* in childhood. Twenty-eight gave a definite age of later syphilitic infection as follows: 5 between 15 and 19 years, 14 between 20 and 29 years, 5 between 30 and 39 years, 2 between 40 and 49 years, and 2 between 50 and 59 years. Twenty-two did not state the age of infection, 9 were uncertain and 42 said they did not have an infection.

Of the 22 who did not state the age of infection, the Kahn test was not carried out on 3, and the reaction was doubtful for 1 and positive for 18. Of the 9 who were uncertain about infection, the Kahn reaction of the blood was negative for 3 and positive for 6. Of the 42 who denied syphilitic infection, the Kahn test was not carried out on 3, and the reaction was negative for 8 and positive for 31.

Anamnesis of 454 Syphilitic Bedouins: Of the 454 seminomadic syphilitic men, 287 (63 per cent) said they had had *bejel* in childhood. One hundred and nine (24 per cent) gave a later age of infection as follows: 15 between 15 and 19 years, 50 between 20 and 29 years, 33 between 30 and 39 years, 7 between 40 and 49 years, 2 between 50 and 59 years and 2 between 60 and 69 years. Ten did not state the age of infection, 20 were uncertain and 28 said they did not have infection.

Of the 10 who did not state the age of infection, the Kahn reaction of the blood was negative for 1, doubtful for 1 and positive for 8. Of the 20 who were uncertain about infection, the Kahn test was not carried out on 3, and the reaction was negative for 5, doubtful for 1 and positive for 11. Of the 28 who said they did not have infection, the Kahn test was not carried out in 4, and the reaction was negative for 6 and positive for 18. One of the patients with a negative Kahn reaction of the blood had a positive reaction of the cerebrospinal fluid.

Thus, of 50 syphilitic Christian men, 25 (50 per cent) gave a history of infection; of 156 syphilitic townsmen, 105 (67 per cent) gave a history of infection, and of 454 syphilitic Bedouins, 406 (90 per cent) gave a history of infection. In other words, only half the Christians admitted having syphilitic infection, while two thirds of the townsmen and nine tenths of the Bedouins did so.

Reactions of the Blood of 660 Syphilitic Men:[4] Of the 50 syphilitic Christians there was a record of Kahn reactions of the blood for 47. Of this number, 9 had a negative reaction (all doubtful reactions are counted as negative), but 1 with a negative reaction of the blood had a positive reaction of the spinal fluid. This makes a corrected total of 8 (17 per cent) with a negative and 39 (83 per cent) with a positive reaction. The positive reactions were distributed as follows: 9, 1+; 16, 2+; 6, 3+; 7, 4+, and 1 (cerebrospinal fluid), 1+.

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4. All the serologic work hitherto done, as well as that reported on here, has been carried out according to the Kahn method in the laboratory of the clinic. Through the courtesy of Bedouins of the American University of Beirut Medical School, a series of specimens of serum is also being tested in the laboratory of the American Hospital in Beirut by both the Kahn and the Kolmer-Wassermann methods. The results in the two laboratories and with the two types of reaction show close parallelism and will be reported on subsequently in full.

Of the 156 syphilitic townsmen, there was a record of the Kahn reaction of the blood for 143. Of this number, 27 (19 per cent) had a negative reaction, and 116 (81 per cent), a positive reaction. The positive reactions were distributed as follows: 48, 1+; 41, 2+; 20, 3+, and 7, 4+.

Of the 454 syphilitic Bedouins, there was a record of the Kahn reaction of the blood for 384. Of this number, 67 had a negative reaction, but 2 of these had a positive reaction of the spinal fluid. This makes a corrected total of 65 (17 per cent) with a negative and 319 (83 per cent) with a positive reaction. The positive reactions were distributed as follows: 98, 1+; 107, 2+; 63, 3+; 49, 4+; 1 (cerebrospinal fluid), 1+, and 1 (cerebrospinal fluid), 2+.

Thus, the Kahn test was carried out on 574 of the 660 syphilitic men. Approximately 83 per cent of the whole series and 83 per cent of each of the constituent groups had a positive reaction. This is a comparatively high percentage of positive reactions, as two factors are neglected, both of which have a tendency to

TABLE 1.—*Syphilitic Lesions and History of Exposure to Venereal Infection in 660 Syphilitic Men*

| | 50 Christians (franghi) | 156 Townsmen (franghi or bejel) | 454 Bedouins (bejel) |
|---|---|---|---|
| Syphilitic lesions | | | |
| Ulcers of mouth, nose and throat | 3 | 28 | 175 |
| Ulcers and eruption of skin | 4 | 18 | 152 |
| Periostitis | 1 | 4 | 45 |
| Arthritis and effusions into the joints | 0 | 3 | 15 |
| Juxta-articular nodules | 0 | 1 | 7 |
| Plantar keratosis | 0 | 0 | 7 |
| Exostoses | 0 | 0 | 2 |
| Orchitis | 2 | 5 | 8 |
| Penile ulcers | 0 | 1 | 6 |
| Chancres | 21 | 15 | 0 |
| Buboes | 3 | 3 | 1 |
| Tabes dorsalis | 1 | 2 | 0 |
| Hemiplegia | 0 | 1 | 0 |
| History | | | |
| Habitual venereal exposure | 12 | 12 | 0 |
| Acute or chronic gonorrhea | 10 | 10 | 0 |

reduce the percentage of positive reactions: First, some of the patients, especially those of the Christian group, had had treatment for syphilis before the blood was tested. Second, many of the patients, especially those of the Bedouin group, had a latent infection dating back decades to a childhood infection.

Syphilitic Lesions and Venereal History of 660 Males: In table 1 are indicated the types of lesions found in 660 syphilitic males and also significant facts relating to exposure to venereal disease. The table is divided into three parts, showing the lesions in Christians, townsmen and Bedouins. It will be noted that ulcers of the mouth, nose, throat and skin and lesions of the periosteum were much more common among Bedouins and that chancres, gonorrhea and a history of exposure were confined to the Christians and townsmen.

It will be noted that of the 660 syphilitic males there were only 36 with initial lesions, i. e., chancres, and that these appeared in 21 of the 50 Christians and in 15 of the 156 townsmen. None of the 454 Bedouins presented a chancre. The penile ulcers found in 6 Bedouins were late lesions. In an experience of ten years I have never seen a chancre on a Bedouin subject, using the word in the usually accepted sense of a genital initial lesion.

Summary: To sum up this section, 40 per cent of the men admitted to the clinic were syphilitic: 12 per cent of the adult male Christians, 21 per cent of the adult townsmen and 66 per cent of the adult male Bedouins. In a series of syphilitic males, 7.5 per cent were Christians, 23.5 per cent were townsmen and 69 per cent were Bedouins. In the same series, infection with syphilis was acknowledged by only half of the Christians, by two thirds of the townsmen and by nine tenths of the Bedouins. The Christians contracted the disease venereally; the townsmen, either venereally or nonvenereally, and the Bedouins nonvenereally. Two thirds of the Bedouins acquired syphilis in childhood. A history of repeated venereal exposure and of gonorrhea was obtained only from Christians and townsmen. Genital chancres were found only on Christians and townsmen. Syphilitic lesions of Bedouin men were mostly late manifestations of the disease, in the mouth, nose and throat, on the skin and in the bones and joints.

2. *Gonorrhea Among 7,000 New Patients.*—One might disregard the foregoing sociologic and clinical facts, which support the Bedouins' own statement that sexual promiscuity does not enter into the propagation of *bejel,* and—taking their own admission that most of them contract *bejel* in childhood—one might argue that the absence of chancres in Bedouin men was due merely to the early and thorough dissemination of the spyhilitic virus among the men and women throughout the community. The absence of chancres under such conditions would not rule out the possibility of sexual promiscuity.

A control which helps to settle this point is found in gonorrhea, an independent venereal disease that elsewhere runs parallel to the distribution of venereal syphilis. Crucial evidence that sexual promiscuity does not exist among the Bedouins is furnished by the statistics on gonorrhea, a disease which is probably an even clearer index of promiscuity than syphilis.

The records of 7,000 consecutive new patients admitted to the clinic showed 149 cases of gonorrhea, with 98 in men and 51 in women. In 72 of these cases smears were positive for the gonococcus; in the rest the smears were negative or there was no bacteriologic examination, the diagnosis being based on clinical symptoms and a history of exposure to venereal disease.

Of the 98 males, 55 were Christians, 41 were townsmen and 2 were nomad Bedouins, for 1 of whom the diagnosis was doubtful (the other had been a soldier and admitted frequent exposure to venereal disease through intercourse). Of the 51 women, 30 were Christians and 21 were townswomen.

The records of the clinic do not show any Bedouin woman with gonorrhea or with any of the sequelae of gonorrhea. Dr. Susan Crosley [5] stated that in five years of gynecological work in the clinic, during which she examined hundreds of Bedouin women, she had not seen an instance of gonorrheal cervicitis, pelvic inflammation or any of the other conditions associated with this disease in women.

5. Personal communication to the author.

Ninety-six of the 98 men with gonorrhea were Christians and townsmen. Two were nomads of the eastern desert, of whom 1 was a soldier and might well be reckoned with the townsmen. The series did not contain any Bedouin villagers. In my experience of examining perhaps 2,000 Bedouin men I have never seen one with gonorrheal urethritis, gonorrheal orchitis, epididymitis, stricture or other manifestation of gonorrheal infection in the male. The clinical examination of these men and women has been too thorough to permit the assumption that gonorrheal infection, if present, would be overlooked, even though not included in the patient's complaints.

The conclusion is inescapable that the Bedouin community is free from gonorrheal infection, a fact that strongly supports the assumption that the Bedouins do not indulge in sexual promiscuity.

3. *Bejel, a Childhood Disease of the Whole Community.*—Up to this point I have been presenting the evidence that promiscuity and

TABLE 2.—*Bedouins' Statements as to Time of Infection with Bejel (1,000 Cases)*

| Time of Infection, Years | Men | | Women | | Children | | Total |
|---|---|---|---|---|---|---|---|
| 0 to 14 | | 219 | | 236 | | 103 | 558 |
| 15 to 19 | | 18 | | 17 | | 0 | 35 |
| 20 to 29 | | 25 | | 33 | | 0 | 58 |
| 30 to 39 | | 12 | | 13 | | 0 | 25 |
| 40 to 49 | | 7 | | 5 | | 0 | 12 |
| 50 to 59 | | 4 | | 0 | | 0 | 4 |
| 60 to 69 | | 3 | | 2 | | 0 | 5 |
| Uncertain | | 13 | | 23 | | 4 | 40 |
| Syphilitic infection denied | | 59 | | 68 | | 89 | 216 |
| Kahn reaction negative | 30 | | 33 | | 25 | 88 | |
| Kahn reaction positive | 16 | | 11 | | 9 | 36 | |
| No test | 13 | | 24 | | 55 | 92 | |
| Total | | 360 | | 397 | | 196 | 953 |
| Blank records | | | | | | | 47 |
| Total | | | | | | | 1,000 |

venereal diseases do not exist among the Bedouin villagers and therefore that syphilis (*bejel*) as it exists among them is not venereal. The negative statement is not sufficient, however, and I shall indicate in this concluding section what seems to be the true nature and course of *bejel.*

Time of Life When *Bejel* Is Contracted: In a case of venereal syphilis the element of shame clouds the patient's anamnesis; the Bedouins, on the contrary, readily acknowledge previous infection with *bejel,* and their statements have a high degree of accuracy, as shown by a check with their reactions to the Kahn test.[6]

In the first series of 177 syphilitic Bedouins,[1c] 80 (45 per cent) said they had had *bejel* in childhood and 46 (26 per cent) named a later specific date of infection.

In a series of 162 apparently healthy Bedouin workmen,[1d] 91 (56 per cent) said they had had *bejel* in childhood and 41 (25 per cent) named a later specific date of infection.

In a series of 62 Bedouin patients [1d] examined at a distance of 60 miles (82.2 kilometers) from Der-ez-Zor, 29 (45 per cent) said they had had *bejel* in childhood and 19 (30 per cent) named a later specific date.

6. Hudson.[1c] Hudson and Young.[1d]

In the anamnesis of the 454 syphilitic Bedouin men mentioned in an earlier part of this paper 287 (63 per cent) said they had had *bejel* in childhood and 109 (24 per cent) named a later specific date of infection.

The largest series of such statements is summarized in table 2. Between April 4, 1933, and March 27, 1935, 2,307 patients of all kinds were admitted to the clinic, of whom exactly 1,000 were Bedouins of all ages. Of these 1,000 Bedouins the patient's statement in regard to infection with *bejel* is lacking for 47. Of those whose statements were recorded, 360 were men; 397, women, and 196, boys and girls aged from new-born infants to 14 years.

Of the 360 men, 219 (61 per cent) said they had had *bejel* in childhood, and 69 (19 per cent) named a later specific date. Only 26 had the first infection with *bejel* after the age of 30, and 13 were uncertain. Fifty-nine stated they did not have syphilitic infection; 16 of these had a positive Kahn reaction.

Of the 397 women, 236 (59 per cent) said they had had *bejel* in childhood, and 70 (18 per cent) named a later specific date. Only 20 had the first infection with *bejel* after the age of 30. Twenty-three were uncertain. Sixty-eight stated they did not have syphilitic infection; 11 of these had a positive Kahn reaction.

Of the 196 children, 103 (52 per cent) were said to have had *bejel*, either in infancy or in earlier childhood, and 4 were uncertain. Eighty-nine stated they did not have syphilitic infection; 9 of these had a positive Kahn reaction.

For the whole series of 1,000, including patients whose statements were lacking and children who had not yet had *bejel*, 55.8 per cent had had *bejel* in childhood and 13.9 per cent named a later specific date of infection, 4 per cent were uncertain and 21.6 per cent stated they did not have infection.

There were 757 patients, male and female, 15 years and older, of whom 455 (60.1 per cent) had had *bejel* in childhood; 139 (18.4 per cent) others had had *bejel* at a later specific time, making a total of 594 (78.5 per cent) who acknowledged syphilitic infection. Only 46 (6 per cent) had had the initial infection after the age of 30.

The Kahn reactions for those who said they did not have infection supported the patients' statements in 2 cases of 3 among the men and in 3 cases of 4 among the women and children.

Thus, in a Bedouin population in which over 60 per cent had a positive Kahn reaction,[1d] 60 per cent of a large series said they had had *bejel* in childhood and another 20 per cent said they had had it at a later specified date; only 6 per cent said they contracted it after the age of 30.

This is evidence not only that *bejel* among the Bedouins is not a venereal disease but that it has a community-wide distribution and that it is usually acquired in childhood.

*Clinical Course and Apparent Epidemiology of Bejel.*[7]—The Bedouin child is not congenitally syphilitic, though both parents have usually had *bejel*. It may be that the child carries over from birth a state of allergy to the syphilitic virus, which perhaps accounts for the par-

7. This summary is based on experience with the disease in the clinic. The underlying clinical and laboratory findings are to be found in the papers already published and in preparation.

ticular way that he reacts to the disease when he contracts it. This point needs to be investigated by the use of the cutaneous test with organic luetin in children who have not yet had *bejel.*

At some time in early life the Bedouin child contracts *bejel,* the source of the infection being some other child in the acute stage of the disease. As this stage lasts several months and does not incapacitate the subject and as the children of the village play together, it is easy to see how few children escape contact with the disease.

In humid, tropical countries where vegetation is luxurious, wounds and scratches are common and heal slowly, offering suitable sites for the implantation of infection by contact or through the medium of the multitude of insects. These climatological features account for the superficial position of initial spirochetal lesions in the tropics. In contrast, the region of Deir-ez-Zor is a dry steppe or desert, with an annual rainfall of about 5 inches (12.7 cm.) and with little vegetation. Cutaneous wounds and abrasions are therefore rare and tend to heal promptly without suppuration when they do occur. This may account for the absence of initial lesions on the skin in *bejel,* the mother sore. Such lesions are never recognized or indicated by the patient with *bejel.*

On the other hand, the Bedouin method of supplying drinking water for the family offers ample opportunity for the transfer of spirochetes and probably determines the usual site of the earliest lesions of *bejel.* The women carry up from the river skins of water, which are laid in the darkened huts. A small bowl without a handle is used to drink from, and every one has recourse to this bowl many times a day. The domestic fly, the louse and the flea are the three ubiquitous insects of the Bedouin hut or tent, and they may also play some part as vectors of the virus. The legions of flies in particular may carry infection from lip to lip. The adults frequently fondle and kiss the children. Altogether, it is not surprising that oral lesions are usually the first to appear in *bejel.*

The child with *bejel* does not usually show much constitutional change, and the Bedouins say that children never die of *bejel.* The lesions on the mucous membrane of the oral cavity are grayish patches with desquamation but with no ulceration. The saliva and scrapings of the lesions swarm with spirochetes of the morphology and characteristic motility of Spirochaeta pallida.[8] About the same time the patches occur in the mouth papules appear about the genitalia and in the folds of the skin. Sexual intercourse does not determine this location.

8. Dark-field studies of lesions of *bejel* are now in progress. The special objective and lamp for this purpose were provided by a grant made by the Ella Sachs Plotz Foundation, and the special condenser was the gift of friends in Collingswood, N. J.

Papules are most common and most succulent where there is warmth and moisture. Other papules may appear on the trunk, usually arranged in circinate configurations. Spirochetes are easily demonstrable on the moist papules. The process in the oral cavity may extend to the bones of the nose, producing softening and collapse of the nasal bridge. Less often, there are tenderness and swelling of the epiphyses and periosteum of the long bones.

In the course of a year these lesions disappear, leaving an apparently healthy child. Few of the Bedouin children are ever presented to a physician, as the Bedouins recognize the infection as inevitable and self-limiting. Few receive curative treatment, though an injection or two of an arsenical or of a bismuth preparation will abolish the lesions.

The child with *bejel* always has a strongly positive Kahn reaction of the blood, which remains positive for many years, though as he grows up he may present no further symptoms of the disease. At some time later, however, in the second, third, fourth, fifth, sixth or even seventh decade of life, there may develop an ulcer of the pharynx, an ulcer or eruptive patch on the skin or a gumma of the nasal bones or one of the long bones. Or the subject may merely complain of aching and throbbing in all the bones of the body.

When some of these late lesions break down one may find spirochetes, but there is usually an enormous admixture of secondarily infecting organisms. Again, 1 or 2 Gm. of an arsenical or 1 Gm. of a bismuth preparation will usually suffice to abolish the lesion. The interval between the first infection with *bejel* and the appearance of these late lesions is usually so great that the Bedouins have not learned to connect them as being due to the same cause.

There is absence of serious damage to the cardiovascular system, the nervous system or viscera.[1e] The rate of miscarriage is much lower than that found in women with venereally contracted syphilis.[9]

If a man has escaped the infection in childhood and subsequently marries, he does not contract syphilis from his wife, though she probably had *bejel* in childhood. But he is almost sure to become infected at some time in his life by contact with some child, perhaps his own, who has open lesions on the mouth. Many of the adult patients with *bejel* give this history spontaneously. When the adult contracts the disease the lesions on the mouth develop first, and then the lesions on the skin and other parts of the body, in the same sequence as *bejel* in the child. In the adult, however, the early lesions often have a tendency to pass directly into the late lesions without an interval of time.

9. These aspects of *bejel* will be considered separately in subsequent papers.

The previously reported studies [6] of the reaction to the Kahn test in the Bedouins showed the same percentage of positive reactions in men and in women of all age groups. The percentage is about 60, whether in patients in the clinic or in presumably healthy persons who are not patients.

The foregoing clinical résumé of *bejel* shows that it is a disease of the whole Bedouin community, resembling a childhood exanthem in its epidemic course.

## COMMENT

In the valley of the Middle Euphrates River, within the territory of the Syrian mandate, is a region isolated from outside contacts, with a population of 200,000, comprised of 5,000 Christians, 45,000 Arab townsmen and 150,000 seminomad Bedouin villagers.

About 1,400 general new patients per year are admitted to the Deir-ez-Zor Clinic, of whom 24 per cent are Christians, 35 per cent townspeople and 41 per cent Bedouins. The three groups are distinct socially and differ in their relation to sexual promiscuity and venereal diseases.

Members of the general Christian population, with an incidence of syphilis of less than 10 per cent (mostly the young men), have venereal syphilis, called *franghi.* Men and women of the Christian group have gonorrhea.

Members of the general Bedouin population, with an incidence of syphilis of over 75 per cent, suffer from nonvenereal syphilis called *bejel,* a disease of the whole community, affecting persons of all ages and both sexes. Neither Bedouin men nor Bedouin women have gonorrhea.

The general town population, with an incidence of syphilis of about 25 per cent, is an intermediate group, some having *franghi,* if they have contracted syphilis venereally in adult life, and others having *bejel,* if they contracted the disease nonvenereally in childhood. Gonorrhea is found in both sexes among the townspeople.

In other words, in this small geographic area syphilis exists in two forms, (1) the purely venereal type and (2) the purely nonvenereal type. The definiteness of these two forms of syphilis in such close physical proximity is linked with and explained by peculiar economic and social factors and is probably not to be duplicated elsewhere.

If this region is opened by roads, commerce and contacts with the outside world, as may indeed happen, the distinctiveness of the three social groups is bound to fade and with it the distinctiveness of the two forms of syphilis.

The present freedom of the Bedouins from promiscuous habits and from venereal diseases is bound to be lost as their customs break down in contact with the forces of civilization. A rise in the economic level,

a more ready access to the towns, a growth of individualism and a loosening of the tribal bonds would all tend to the introduction of promiscuity and venereal diseases. These Bedouins are by no means paragons of sexual morality. Only the combination of isolation, straitened economic conditions and a rigid tribal sexual code maintains their present freedom from venereal diseases.

The process of disintegration is already apparent in their cousins, the townspeople, and the similar disintegration of the villagers will probably not long be delayed. Hewer [10] attributed the introduction among the Arabs of the Sudan of gonorrhea (which is now universal) and syphilis (which now exists "side by side with yaws") to the breaking down of local social barriers and the consequent dissemination of imported diseases.

The juxtaposition of *franghi* and *bejel* in the region of Deir-ez-Zor brings into focus the fact that the character of syphilis varies with the particular epidemiology of its locale. *Bejel* operates under the epidemiologic triad of (1) widespread community infection, (2) childhood acquisition and (3) general lack of treatment. *Franghi* occurs in a restricted adult portion of the towns and is definitely venereal. *Franghi* and *bejel* have in common the etiologic agent, the reaction of the blood and the favorable response to the same drugs, but the clinical courses in the human subject are strikingly different.

That *bejel* is a spirochetosis is beyond doubt. The uniformly positive reactions of the blood and the constant presence of a spirochete with the morphology and motility of S. pallida in dark-field preparations from the early open lesions settle this point. It is possible that *bejel* is the manifestation of a peculiar strain of S. pallida. This point can be settled only by experimentation on animals.

The parallelism of *bejel* and yaws is impressive. Yaws operates under the same epidemiological triad as *bejel*. But to call *bejel* yaws would be to create more difficulties than would be solved. The clinical course of the two diseases, though remarkably similar, is not identical. Stannus said: [11] "I should like to emphasize the fact that yaws is strictly a tropical disease . . . limited by Cancer and Capricorn." Stitt [12] made the same statement and added that it is found almost exclusively in the dark-skinned races. Deir-ez-Zor is over 10 degrees north of the Tropic of Cancer, and the Bedouins are Semites, not related to the

10. Hewer, T. F.: Some Observations on Yaws and Syphilis in the Southern Sudan, Tr. Roy. Soc. Trop. Med. & Hyg. **27**:593, 1934.

11. Stannus, H. S.: Yaws and Syphilis, Brit. J. Ven. Dis. **4**:55, 1928.

12. Stitt, E. R.: The Diagnostics and Treatment of Tropical Diseases, ed. 5, Philadelphia, P. Blakiston's Son & Co., 1929.

dark-skinned races. It would be hard to picture a pocket of yaws developing in this area so far from the regions in which yaws is endemic.

It would be gratuitous to introduce yaws as an explanation of the character of *bejel* or even to bring in the concept of a special strain of virus when a simpler explanation is at hand, one that meets the demands of the equation adequately. It seems to my associates and me that such an explanation does exist and that the true explanation may be stated as follows: The dissimilarity in the epidemiology of *bejel* and *franghi* accounts for the difference between these forms of spirochetosis, and the similarity in the epidemiology of *bejel* and yaws accounts for the likeness of these two forms of spirochetosis.

It has been assumed in the literature that there is such a disease entity as acquired nonvenereal syphilis of childhood, such as *bejel.* Stannus said: [11]

> . . . Infection with syphilis may, and does, take place in childhood in some native communities where the disease is widespread, and the primary lesion is then commonly extragenital. . . . Many observers, writing of syphilis among native children, appear to believe they are dealing with the inherited disease, and note with surprise the absence of some of the common stigmata, whereas I believe they are dealing with late syphilis, the disease having been contracted in infancy, and it must never be lost sight of that tertiary lesions often appear early. . . . For comparison with the pathology of yaws, I would suggest that a study of the pathology of syphilis in a native race be undertaken. There is a good deal of evidence that the manifestations of that disease among indigenous natives differ somewhat from those seen in Europeans, evidences that the reaction to infection tends to differ in the two cases.

In a previous paper [1c] from the Deir-ez-Zor Clinic the following statement was made:

> Most writers and reviewers of literature of tropical medicine are taken up with yaws, and with its differentiation from syphilis, to the exclusion of the larger subject as to the nature of syphilis itself in primitive peoples. Advocates of the duality of the two diseases overlook the fact that their strongest argument would be to delineate the symptoms and pathology of this pandemic syphilis which they state exists as a distinct disease side by side with yaws in tropical countries.

In spite of the importance and the desirability of a thorough study of syphilis during childhood acquired in a native people, it has never been made. In fact, Blacklock said: [13] "What the course of acquired syphilis of early childhood may be is, and must remain, largely problematical, and a matter of speculation." Why does he despair of exact information about this form of syphilis? Why has not some one made the study of the pathology of "syphilis in a native race" which Stannus suggested?

13. Blacklock, D. B.: Yaws and Syphilis, Ann. Trop. Med. **26**:423, 1932.

This has not been done because syphilis acquired in childhood occurs almost exclusively in those parts of the world where endemic yaws is also found. The confusion of the two diseases is too great to be unraveled. In a given case the question always arises: Is it syphilis of childhood, is it yaws or may it be a mixed infection? Hewer [10] in the Sudan said that it was his original plan to investigate parallel series of cases of syphilis and yaws and to compare the results, but unfortunately it soon became clear that a differential diagnosis based on the usually accepted criteria was impossible; a definite decision could be made only by exercising one's personal prejudice. He therefore deemed it more "honest" to describe the manifestations in the whole group collectively.

Turner and Saunders [14] in Jamaica attempted the task of weeding out the cases of "early syphilis" from those of "early yaws," and drew tentative conclusions from 12 cases of the former which they found among thousands of the latter. The difficulties in this method of comparing the two diseases are obviously enormous.

In the discussion of yaws and syphilis the question constantly arises: What would syphilis be like if it were propagated under epidemiological conditions similar to those of yaws? As long as the Bedouins of the Euphrates Valley have *bejel* free from the venereal factor, this question can be answered, and the course of the acquired syphilis of early childhood need not remain "largely problematical, and a matter of speculation."

Blacklock [13] said: ". . . The clue to the solution of the yaws and syphilis question appears to lie in syphilis as it occurs in the natives of the tropics. . . . The effects of extragenitally acquired syphilis in the native children of rural areas of the tropics are those about which we chiefly require information."

It seems probable that a study of *bejel*, the nonvenereal form of syphilis acquired in childhood among the homogeneous and isolated group of 150,000 Bedouins of the Euphrates River valley, in whom yaws does not occur, can supply the required information and furnish the required clue.

## SUMMARY

The settled population of the Middle Euphrates valley comprises 5,000 Christians, 45,000 townspeople and 150,000 Bedouin villagers. Physical barriers separate the people of this region from the outside world, and social and economic barriers separate the three groups from each other.

14. Turner, T. B., and Saunders, G. M.: Report of the Jamaica Yaws Commission for 1933.

Promiscuous sexual intercourse and venereal diseases are found among the Christians to a degree comparable to that in the West. Gonorrhea, venereal syphilis (*franghi*) and nonvenereal syphilis (*bejel*) are all found among the townspeople. Neither promiscuity nor venereal diseases are found among the Bedouins.

Syphilis among the Bedouins is called *bejel*. It is a community infection, and few persons escape the disease. The majority contract *bejel* in childhood, and the reservoir of open lesions is among the children.

*Bejel* is syphilis without a venereal factor, and it presents the unusual feature of nonvenereal syphilis in a large native population in which the disease of yaws can be excluded.

The direct comparison of yaws and syphilis has always been clouded by the fact that venereal epidemiology is either assumed in, or at any rate cannot be eliminated from, syphilis.

*Bejel*, however, is a form of syphilis that has the same epidemiology as yaws in that sexual intercourse is not concerned in its propagation. *Bejel* also resembles yaws in its widespread distribution in the community, its acquisition in childhood and the general lack of treatment.

A study of *bejel* therefore might throw light on the relationship of syphilis and yaws, since it would enable one to make direct comparison of the two spirochetoses operating under identical epidemiological conditions.

Such a study will remain possible only as long as *bejel* retains its present freedom from the venereal factor. Present-day tendencies are all against *bejel* retaining its nonvenereal character.

A scientific control in the study of *bejel* is afforded by *franghi*, the venereal form of syphilis existing in the towns of the same region.

To label as syphilis all venereally acquired spirochetosis and as yaws all nonvenereal spirochetosis is unscientific. The use of a history of venereal infection or genital lesions as points of differentiation between syphilis and yaws is not justified.

# EVALUATION OF THE PHYTOPHARMACOLOGIC TEST OF PELS AND MACHT

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In an effort to evaluate the phytopharmacologic test which Macht and Pels [1] described as a diagnostic measure in cases of pemphigus, we undertook several experiments. (Macht [2] has been reporting on the test for the past ten years and found it of diagnostic value in cases of pernicious anemia.) The test is based on a comparison of the amount of growth of roots of Lupinus albus Hartwegii seedlings grown in a plant nutrient solution (Shive [3]) to which 1 per cent of blood serum of a patient has been added with the amount of growth of similar seedlings in plant nutrient solution which contains 1 per cent of blood serum from normal subjects. The amount of growth retardation is taken to indicate the toxicity present in the patient's serum.

## METHOD

The method used by Pels and Macht, as reported in 1931, is as follows:

1. Seeds of Lupinus albus Hartwegii are soaked overnight in tap water.
2. The soaked seeds are sprouted in ground sphagnum moss (previously washed and dried) to which water has been added to approximately 80 per cent of the weight of the moss.
3. After forty-eight hours in sphagnum, hardy seedlings with roots of approximately equal length are selected, and the length of the roots is measured in millimeters.
4. The roots are then immersed in a 1 per cent solution of the serum to be tested and kept at room temperature (22 C.) for twenty-four hours.
5. After twenty-four hours the roots are measured again, and the average growth is determined.

In our experiments the method just outlined was followed at first, but in order that additional information might be obtained, certain modifications of the proce-

From the Pittsburgh Skin and Cancer Foundation.

1. Macht, D. I., and Pels, I. R.: Phytopharmacology of Pemphigus and Other Dermatoses: Additional Studies, Arch. Dermat. & Syph. **23**:601 (April) 1931.

2. Macht, D. I.: Phytopharmacological Study of Pernicious Anemia, J. Pharmacol. & Exper. Therap. **29**:461, 1926.

3. Shive, John W.: A Study of Physiological Balance in Nutrient Media, Physiol. Researches **1**:327, 1915.

dure were carried out in an attempt to vary the amount of growth retardation and also to determine, if possible, the cause of it.

We are using several terms in this publication which we believe need definitions to clarify them:

1. The mean (M), as used here, is the sum of all the measurements of a group of seedlings divided by their number.

2. The standard deviation (S.D.) is a measure of the variability or lack of uniformity of the length of rootlets in a group of seedlings. The mathematical formula used for estimating it is $\frac{\Sigma d^2}{n}$, in which $n$ equals the number of seedlings in a group and $d$ equals the deviation or difference of each seedling from the mean. To determine the sum ($\Sigma$) $d^2$ one calculates the difference of each measurement from the mean, squares these differences and adds the squares. The mean $\pm$ 3 S.D. has been shown statistically to include 99 per cent of all other seedlings of similar type and treatment.

3. The standard error (S.E.) of the mean is a measure of the reliability of the mean. This is obtained when the standard deviation of any group is divided by the square root of the number of seedlings. The standard error of a mean measures the chances that any additional seedling of identical derivation has of being accurately represented by that mean. There is a 1:1 chance that the measure of an additional seedling would lie within the quantities M $\pm$ S.E.; there are four and two-tenths chances that the seedling would lie within to one that it would lie without the values M $\pm$ 2 S.E. The chances are 21:1 that the seedling will lie within the values M $\pm$ 3 S.E.

4. The plant nutrient solution will be spoken of as the Shive solution; its composition is as follows:

| | Cc. |
|---|---|
| 0.5 molar solution of calcium nitrate | 10.4 |
| 0.5 molar solution of magnesium sulfate | 30.0 |
| 0.5 molar solution of potassium di-hydrogen sulfate | 36.0 |
| Distilled water to make | 1,000.0 |

## REPORT OF THE EXPERIMENTS

*Methods Used in Sprouting Seedlings.*—Pels and Macht in their experiments sprouted the seedlings in washed ground sphagnum. This we did also. Numerous tests have shown that seedlings sprouted in sphagnum and then grown in Shive's solution for twenty-four hours do not grow as long as seedlings sprouted on moistened filter paper in large sterilized Petri dishes and then grown in the Shive solution. Three hundred and thirty Lupinus seedlings sprouted in sphagnum when grown for twenty-four hours in the Shive solution had a mean growth of 7.73 mm. $\pm$ 0.0872 S.E., as compared to a mean growth of 11.26 mm. $\pm$ 0.1373 S.E. for 464 plants sprouted on Petri dishes and grown in the Shive solution for twenty-four hours afterward. Sprouting in sphagnum conditions a lower rate of subsequent growth than sprouting in Petri dishes.

*Comparison of Growth of Seedlings of Different Plants.*—The seedlings of plants belonging to widely separated groups were tested to observe their behavior and to find, if possible, a more sensitive seedling than that of Lupinus.

(*a*) Seedlings of Raphanus (radish) were found to be very small and supersensitive to all sorts of laboratory conditions; many of them died and thus were found unsatisfactory for experimental handling.

(*b*) Fagopyrum (buckwheat) sprouted well in the Petri dish chamber, but the roots of the seedlings when immersed sent out many side rootlets which made accurate measurement very difficult. These plants, too, were often killed in the serum solution and therefore were unsatisfactory for experimental handling.

(*c*) Allium (onion) seedlings were found to be too small as well as extremely variable in their reactions and therefore were discarded.

(*d*) It occurred to one of us (R. J. G.) that a rootlet normally accustomed to a hydrophytic environment might be best adapted to immersion in solutions. Therefore, Salix (swamp willow) cuttings were immersed in tap water. After fifteen days the rootlets were long enough for measurement and therefore for use. During the twenty-four hour test period one hundred and eighteen rootlets grew to the mean value of 0.788 mm., an amount too small for experimental purposes, and this plant was discarded.

(*e*) Seedlings of Zea mays ("white rice" popcorn) simulated those of Lupinus very closely. Even a larger percentage of the sprouted Zea seeds could be used than those of Lupinus because they are less affected by experimental treatment. Consequently, these seedlings were added, we believe advantageously, as test plants in many experiments.

*Variation in Growth of Seedlings of Lupinus.*—The weakest point of the phytopharmacologic test lies in the fact that any group of plant seedlings of any particular plant species will show a striking variation in the rate of its growth. Two hundred and eight seeds of Lupinus were soaked in tap water for ten hours and then put into sterile Petri dishes on moistened filter paper. These were kept at room temperature for forty-seven hours and then measured. Of these seeds, forty-one did not sprout; twenty-two grew from 1 to 3 mm., nineteen from 4 to 6 cm., thirty-four from 7 to 9 mm., twenty-nine from 10 to 12 mm., twenty-five from 13 to 15 mm., fifteen from 16 to 18 mm., and twenty from 19 to 21 mm., and three grew 23, 26 and 27 mm., respectively. The mean amount of growth was 11.01 mm., the S.D. 5.9448 and the S.E. $\pm 0.46$. The range of growth was from 1 to 27 mm. This shows that similar seeds vary widely in their growth potentialities.

Further proof of this point is presented by a study of the amount of growth of seedlings immersed for twenty-four hours in normal (no demonstrable disease) serum solution. One hundred and ninety Lupinus seedlings were grown in a 1 per cent solution; forty-four grew from 1 to 3 mm., sixty-seven from 4 to 6 mm., forty-six from 7 to 9 mm., twenty-four from 10 to 12 mm. and nine from 13 to 18 mm. The mean growth was 6.28, the S.D. 3.488 and the S.E. $\pm 0.2531$. The variation in the amount of growth was not as great as in the previous experiments. That was because only those seedlings which had attained a medium amount of growth in the Petri dishes were used, and therefore a selection of seedlings had been made. Another factor was the retarding influence of the serum solution itself. However, the range in growth rate was still found to be very great in the individual seedlings. This variability was present in all of our experiments.

At this point two questions arose: 1. Do the seedlings which attain exactly the same amount of growth during their sprouting time in the Petri dish show a highly variable rate of growth when they are grown in a solution later? 2. Does the later rate of growth vary with the rate of growth in the Petri dish? Do seedlings that are 15 mm. in length when taken from the Petri dish grow more slowly in the solution than those which measure 25 to 35 mm. at the end of the sprouting period?

To answer these questions a special experiment was undertaken. A large number of Lupinus seeds were sprouted in Petri dishes. At the end of the sprouting period, that is, in sixty hours, the seedlings which attained root lengths of 15 mm., 20 mm., 25 mm., etc., were selected and immersed in the Shive solution. At the end of twenty-four hours the seedlings were measured. It was found that those measuring 15 mm. at the time of immersion grew from 17 to 35 mm. in length; those measuring 20 mm. at the time of immersion grew from 25 to 40 mm.

Table 1 reports that experiment and shows that the growth of all the groups after a twenty-four hour immersion in the Shive solution ranged from 2 mm. to 20 mm. in spite of the fact that the plants had previously shown a uniform rate of growth. The size of the plant at the end of the sprouting period does not give any indication of what the later growth of it will be. This shows that individual plants vary a great deal in their growth response, and since the rate of growth in the sprouting medium offers no clue as to the growth rate later, it is imperative that a large number of seedlings be used in each test. If a few seedlings only are used, the chances of selecting plants of equal growth potentialities are lessened, and the factor of growth potentiality has a great effect on the mean growth obtained.

TABLE 1.—*Variation in Growth Shown When Plants Are Immersed in a 1 Per Cent "Normal" Serum*

| No. of Seedlings | Kind of Seedlings Used | Length at Immersion | Range in Growth in 24 Hours Measured in Mm. | | | | | Mean Growth |
|---|---|---|---|---|---|---|---|---|
| | | | 2–4 | 5–8 | 9–12 | 13–16 | 17–20 | |
| 11 | Lupinus | 15 | 2 | 1 | 2 | 3 | 3 | 12.27 |
| 17 | Lupinus | 20 | 0 | 4 | 5 | 6 | 2 | 11.76 |
| 9 | Lupinus | 25 | 0 | 4 | 4 | 1 | 0 | 9.00 |
| 17 | Lupinus | 30 | 3 | 0 | 8 | 5 | 1 | 10.88 |
| 12 | Lupinus | 35 | 1 | 2 | 4 | 3 | 2 | 11.50 |
| 5 | Lupinus | 40 | 0 | 1 | 2 | 1 | 1 | 11.80 |

The fact that any information obtained from a small group of seedlings is unreliable can further be proved by the fact that the standard error is always large, showing that the mean is representative of only a small number of plants used and that the measurements obtained for most seedlings were scattered at some distance from the mean. Nineteen Lupinus plants were grown in a 1 per cent serum solution. They had a mean growth of 6.31 mm., a standard error of $\pm 0.5742$, and a range from 5.16 mm. to 7.46 mm. When fifty-seven plants were similarly tested they gave a mean of 6.45 mm., with a standard error of $\pm 0.3513$. The probable location of the mean in this instance was between 5.75 and 7.15, which shows that even with fifty-seven seedlings one can place only an uncertain value on the mean and, further, indicates what a variable factor the rate of plant growth is.

*Effect of Different Percentages of Serum.*—The effect of different percentages of serum added to plant nutrient solution was then studied. The concentrations were 0.5, 1, 5, 25 and 33.3 per cent. When sufficient serum was available, several percentages of the same serum were used, and seedlings of two different plants were imposed in parallel series. Table 2 shows these results and definitely proves that the addition of serum of any type causes a retardation in the growth of the plant and that the greater the concentration the greater the retardation becomes. When the concentration is even as high as 5 per cent, the rate of growth is so markedly

retarded, even by a normal serum, that there is little opportunity for differences in toxicity to become apparent. The difference between the various serums becomes so small as to be unreliable.

*Modification of Pels and Macht Technic.*—The modification in the Pels and Macht technic was carried out in the following manner: After the experiment was concluded and the plants were removed from the serum solution, each seedling was gently blotted with absorbent paper and immersed in the Shive solution for twenty-four hours; then the growth of the plant was measured. Plants grown in the Shive solution grew 11.26 mm. in the first twenty-four hours and 8.91 in the second twenty-four hours, showing a decreased rate of growth during the second period. When plants were first grown in a serum solution and then in the Shive solution, the rate of growth was increased during the second period. After immersion in a 1 per cent serum solution, the later growth ranged around 9 mm., showing that there was a release from the inhibiting agents contained in the serum solution. After immersion in a 5 per cent serum solution the rate of growth in the Shive solution was 7.5 mm., after immersion in a 25 per cent

TABLE 2.—*Effects of Various Concentrations of the Shive Solution*

| Number of Seedlings | Kind of Plant Used | Kind of Solution | Mean Growth in Millimeters | Standard Deviation |
|---|---|---|---|---|
| 330 | Lupinus albus | Shive solution | 11.26±0.1373 | 2.231 |
| 190 | Lupinus albus | 1% "normal" serum | 7.72±0.2531 | 3.488 |
| 253 | Lupinus albus | 1% "pernicious anemia" serum | 6.65±0.1910 | 2.939 |
| 333 | Lupinus albus | 5% "normal" serum | 3.06±0.767 | 1.140 |
| 88 | Lupinus albus | 5% "pernicious anemia" serum | 2.34±0.1682 | 1.581 |
| 520 | Lupinus albus | 5% "carcinoma" serum | 3.84±0.0473 | 1.080 |
| 293 | Lupinus albus | 25% "carcinoma" serum | 2.64±0.0447 | ..... |
| 151 | Lupinus albus | 33.3% "carcinoma" serum | 2.48±0.0333 | 0.410 |
| 198 | Zea Maize (W. R.) | Shive solution only | 9.78±0.3910 | 5.502 |
| 191 | Zea Maize (W. R.) | 1% "pernicious anemia" serum | 4.58±0.2250 | 3.110 |
| 30 | Zea Maize (W. R.) | 5% "pernicious anemia" serum | 2.03±0.2810 | 1.5387 |
| 23 | Zea Maize (W. R.) | 5% "pemphigus" serum | 5.00±0.2920 | 1.979 |
| 425 | Zea Maize (W. R.) | 5% "carcinoma" serum | 3.60±0.0555 | 1.1459 |
| 265 | Zea Maize (W. R.) | 25% "carcinoma" serum | 3.28±0.0450 | 0.7330 |
| 94 | Zea Maize (W. R.) | 33.3% "carcinoma" serum | 1.09±0.0399 | 0.3847 |

serum solution, 6.5 mm., and after immersion in a 33.3 per cent serum solution, 5.55 mm. This shows that the rate of growth increases in the plants which are immersed in a serum of greater concentration, but, of course, this subsequent growth rate is proportional, that is relative, because the growth is lessened.

## CONCLUSIONS

Of a number of plants tested, only Zea mays ("white rice popcorn") was found to react similarly to Lupinus in serum solutions.

Seedlings sprouted in a moist chamber were shown to attain and maintain a more rapid rate of growth than seedlings sprouted in sphagnum or sawdust.

Tremendous variation occurs in rate of plant growth. Tests showed that apparently similar seeds vary widely in their growth potentialities.

It was shown that a mean amount of growth taken from a group of from fifteen to twenty seedlings was a very unreliable mean statistically and that therefore such a mean could not be taken as an adequate index of toxicity of serum.

It was found that plants which were of the same length when immersed in a serum solution showed considerable amount of variation in rate of growth in solution—further evidence of the unreliability of the phytopharmacologic test.

By statistical analysis of the data, it is shown that as the number of seedlings for any test is increased the reliability of the mean increases.

Higher concentrations of serum, 5, 25 and 33.3 per cent, have too severe an effect on the seedlings to be of any test value.

Seedlings that have been immersed in a serum solution for twenty-four hours and then transferred to a normal plant nutrient solution show varying effects of the serum treatment. The effects increase with increase in concentration of the serum solution.

# THERAPEUTIC TIMIDITY

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Of all the members of the animal kingdom the human being is the one that comes most intimately and oftenest in contact with deleterious substances, either as medicines or in the arts and crafts. The deleterious effects of medicines have been recognized for many years. Carolus Rueus, Soc. Jesu, long ago drew attention to the Greek word *pharmacon* (hence our word, pharmacy), which sometimes meant a remedy and sometimes a poison.[1]

The learned, Jesuit commentator on Virgil, Carolus Rueus, stated: *"Pharmacon enim ambigua vox est: et modo remedium modo venenum significat."* (The word *pharmacon* is indeed ambiguous and sometimes means a remedy and sometimes a poison). Rueus, Carolus, in P. Virgilii Maronis Opera, edition by John Cary, London, 1835.

The physician observing the deleterious effects of medicines in his practice runs the danger of becoming overcautious, a disastrous frame of mind. This timidity is often still further exaggerated by the immense mass of literature on industrial medicine wholly occupied with the injuries caused by, and the possible injurious effects of, substances used both in medicine and in the trades.

For example, lead, which is employed as the subacetate, the carbonate or the oleate, constitutes one of the most potent remedies. That lead when ingested or inhaled, whether in solution, as in drinking water, or as a vapor or dust, as in the arts and crafts, may cause plumbism is undoubted, and that lead in its pharmaceutic forms is absorbed when it is applied on the skin there is also no doubt. Sussmann's experiments, to be mentioned later, are here positively conclusive. Therefore, it is more than theoretically possible for plumbism to occur if the medicament is applied over an extensive surface and for a long time. Yet this danger must be remote, and it should not interfere with the employment of such a valuable series of remedies. For example, the bland, unctuous carbonate of lead is an excellent ingredient in an ointment for infantile eczema and is smeared over the face abundantly. As an infant's hands are always wandering over its face and into its mouth, some of the lead compound must be swallowed. Yet lead poisoning from such a source must be very infrequent. The infrequency may be

1. Pharmacal, from the Latin *pharmac-um*, Greek *pharmak-on*—poison or drug (Oxford Dictionary).

due, however, to the free elimination in the feces and urine characteristic of infancy.

Though the actual danger of plumbism may be slight, the dermatologist should consider it, in the first place in estimating the remedy and in the second, and very significantly, to avert any excessive fear of it. Slight, however, as is the danger of inducing plumbism by the application of lead preparations on the skin, one does occasionally meet with physicians willing to abandon them or to interdict their use, valuable as they are.

If physicians were to abandon all remedies having possible injurious by-effects they would relinquish all their most potent ones, such as arsenic. I shall not readily forget my feelings when, in 1910, in Magdeburg, Germany, I first saw arsphenamine, containing fifty times the lethal dose of arsenic, administered intravenously. It seemed to me more like an embalming procedure than a medical experience. There is no doubt that arsenical dermatitis is a grave and distressing malady; yet who would advise abandoning this wonderful remedy just because it at times acts unfortunately?

Take for example a concrete instance:

> Some time ago a woman consulted me on account of severe lesions of both soles caused by roentgen radiation. There were the usual tender keratoses that caused exquisite pain in walking, and this was so grave as to interfere seriously with her ability to gain her living. Moreover, the lack of exercise and the depressant effect of the pain threatened permanent invalidism. Added to this was the fear of ultimate cancer, which had arrived at the point at which ablation of the sole and skin grafting were seriously considered. Even if it should be successful as a graft, this would be a desperate measure, as the sole is furnished with a special skin, and no other would be efficient. Lead is a soft, unctuous, bland heavy metal and it seemed to be the most appropriate application for the irritable condition present; so ointment of lead oleate was prescribed, together with other measures tending to improve the circulation in the feet, such as massage and bandaging of the legs. The improvement was decided. The patient was soon able to walk and stand without discomfort, and with the absence of pain and with the resumption of exercise her normally good health was restored. Of course some of the keratoses are still present, and the condition of the sole is still that of a roentgen ray burn, but the amelioration is decided.

This case is cited simply to illustrate the subject of the paper. As for the good result achieved, there was nothing wonderful in it. It was only what could be expected. The bland effect of ointment of lead oleate has been known throughout the dermatologic world since Hebra's day.[2] In this case it softened and soothed the painful, tender keratoses, and it would also unite with any free liquid albumin, forming lead

2. Hebra, F., quoted by Richter, Paul: Geschichte der Dermatologie, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1928, vol. 14, pt. 2, p. 61.

albuminate, not at all agreeable to any stray parasite happening to come along to enjoy the degenerated cells.

While I was congratulating myself on the improvement of the condition, a fellow practitioner suggested the probability of constitutional lead poisoning, although no symptoms were present. As I have remarked, lead can be absorbed through the skin, and inferentially may cause toxic symptoms, and in this case lead ointment was applied over a fair extent of surface, subjected to the tepid heat of the foot and each day thoroughly massaged in for hours through the action of walking. If these facts alone were considered it would appear that my colleague was justified in his fears. Against this hypothetic danger was the practical fact that ointment of lead oleate has been used in Germany as a routine treatment for sweating feet in thousands of cases. The interesting experiments of Sussmann previously mentioned cast a welcome light on this question. He found that lead in aqueous solution was absorbed through a square meter of the skin at the rate of 0.1 to 0.2 mg. a day.[3] Incorporation in a salve greatly increased this absorbability, so that 0.5 mg. ($\frac{1}{130}$ grain) was absorbed from 0.1 square meter of skin in twenty-four hours. In this experiment the lead salve was thoroughly rubbed into the skin and then enclosed with a dressing. It was found that the addition of a soap to the ointment still further increased the absorbability.[4]

An important finding, however, was that the sebaceous system furnishes the gateway for this absorption. None passes through the horny layer, no matter how thin it is, and the corkscrew-shaped ducts of the sweat glands are also incapable of absorption. Atrophy of the skin together with its glands, as in scleroderma, commensurately retards absorption. From this one can see that in the case cited there could be no danger of absorption through the soles, as neither the palm nor the sole is furnished with either sebaceous glands or hairs.

On the contrary, it is interesting to note that in some countries, Germany for instance, lead hair dyes are interdicted by law and that in the scalp the pilosebaceous system is extremely highly developed, furnishing the requisite gateway for absorption. Even here, however, the occurrence of plumbism may be rather an assumption deduced from the real danger present in the arts and crafts, and in drinking water delivered through lead pipes, rather than any actual danger from hair dyes.

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3. Perutz, A.: Pharmakologie der Haut, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1930, vol. 5, pt. 1, p. 139.

4. Frey, M. V., and Rein, H.: Physiologie der Haut, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1929, vol. 1, pt. 2, p. 6.

The lesson to be learned from the foregoing comment is that, as physicians are continually dealing with highly toxic substances administered to the most variously sensitive of all animals, they must be continually on their guard against nebulous dangers exaggerated by the abundant literature and often by their own timidity, as they might deprive themselves of the use of, or cause themselves to use lamely and ineffectively, many of their most prized remedies.

# AN ACNEFORM DERMATERGOSIS

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It is only within comparatively recent times that the dermatologist has interested himself in dermatoses associated with or caused by the daily work. With the advent of a more clearly defined appraisal of the words eczema and dermatitis and the more general use of the patch test, dermatologists have necessarily inquired more and more deeply into the innumerable chemical contacts that occur in the day by day lives of their patients. As well as being conversant with dermatology, the dermatologist must have a working knowledge of the manufacture and content of the many new and old compounds that are handled and applied by large numbers of persons. Hence, more and more dermatologists are becoming industrially minded, and it is dawning on them that the solution of some of their industrial difficulties may aid them in a clearer conception of the dermatoses that are encountered in their every-day practice.

Dermatergoses of the acneform type occurring in persons working with chemicals have been repeatedly reported in Europe and discussed as industrial "chlor-acne."[1] This term "chlor-acne" was first used by Herxheimer[2] in 1899 to describe an eruption composed of comedones and small sebaceous pustules that occurred on the arms and faces of workers manufacturing chlorine gas electrolytically, using carbon electrodes. It was natural to assume that the chlorine was the causative agent. Bettman[3] had observed in March 1897 two patients whose skins were diffusely pigmented, dark, rough and dry, in whom were observed small tenacious comedones associated with numerous small follicular abscesses. Subsequently he reported twenty-one additional cases with similar symptoms in workmen who were engaged in cleaning out an acid tower used in the manufacture of hydrochloric acid. At no time did these men come in contact with free chlorine, but numerous loose

Read at the Fifty-Eighth Annual Meeting of the American Dermatological Association, Inc., White Sulphur Springs, W. Va., May 2, 1935.

1. White, Prosser: The Dermatergoses, ed. 4, New York, Paul B. Hoeber, Inc., 1934.
2. Herxheimer: München. med. Wchnschr. **46**:278, 1899.
3. Bettman: Deutsche med. Wchnschr. **27**:437, 1901.

derivatives of coal tar were present. Herxheimer later absolved the chlorine but considered the trouble to be due to various chlorbenzenes.

From time to time other reports of the occurrence of "chlor-acne" have been made in the European journals, each author continuing the error of Herxheimer in assuming that the cause of the condition was chlorine. Some of these reports have been indefinite as to the chemical conditions under which the men worked, except that they were exposed to chlorine gas. However, some authors have suspected various chlorbenzene derivatives, such as hexa-chlorbenzene, hexa-chlorethylene, para-nitrobenzene, perchlornaphthalene ("perna") and probably others.[4] Chlorinated naphthalene was first indicted by Wauer [5] in 1918 as a cause of acneform eruptions of the skin, the condition being called *Pernakrankheit*. His observations were confirmed by Koelsch [6] and recently by Teleky.[7] The latter reported an exhaustive study of a large number of cases occurring in workers engaged in the manufacture of perchlornaphthalene, in which the workers were exposed to fumes of the molten mass and also to the sublimated dusts. In approximately one-half of the workers exposed there developed comedones of a particularly heavy and tenacious type with resultant sebaceous abscesses, containing heavy yellow pus and serum. Considerable improvement resulted when the chlorine content of the mass was decreased from 30 per cent to 8 per cent, and Teleky believed that the disease was due directly to the halogen content of the substance. He mentioned the similarity of the disease to *teeracne* (tar acne) but carried the analogy no further. Individual predisposition appeared to alter receptivity.

Some authors have leaned to the view that the acneform eruption is due to direct external contact with the chemicals (notably Bettman, Hallopeau, Jacquet, Fumouze, Teleky and Koelsch [8]). Others (Lehmann, Herxheimer, Jacobi, Roth and Kobert [9]) considered it to be a

4. Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Welfare, International Labour Office, Boston, World Peace Foundation, 1925, brochures 34 and 285.

5. Wauer, cited in Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Welfare, International Labour Office, Boston, World Peace Foundation, 1925, brochure 34.

6. Koelsch, F., in Ullmann, K.; Oppenheim, M., and Rille, J. H.: Injuries to the Skin, Leipzig, Leopold Voss, 1926, vol. 2, p. 303.

7. Teleky: Klin. Wchnschr. **6**:845 (April 30); 897 (May 7) 1927; **7**:214 (Jan. 29) 1928.

8. Bettman; Hallopeau; Jacquet; Fumouze; Teleky, and Koelsch, cited in Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Welfare, International Labour Office, Boston, World Peace Foundation, 1925, brochure 285.

9. Lehmann; Herxheimer; Jacobi; Roth, and Kobert, cited in Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Welfare, International Labour Office, Boston, World Peace Foundation, 1925, brochure 285.

dermatitis due to the absorption of chemical compounds by the lungs or gastro-intestinal tract, with elimination by way of the sebaceous glands. The majority of writers on the subject, particularly Prosser White,[1] have seemed to feel that chlorine as such has little to do with the formation of the comedones and cysts, and they have repeatedly referred to tar and products of the distillation of tar as the prime causative factors. Prosser White rebuked authors for using the term "chlor-acne" at all and expressed the belief that the process is one of the manifold cutaneous reactions produced by tar and its derivatives.

Recently we have had the opportunity to study, with the full cooperation of the manufacturers, an outbreak of acneform eruption occurring in a group of workers engaged in the manufacture of chlorinated di-phenyl. This study has brought out some points in the production of this unusual dermatosis that we feel are important in solving its exact cause. The following case is typically illustrative of the disease as it occurred in the men working in this plant.

## REPORT OF A CASE

*History.*—O. D., a Negro aged 26, began work in the distillation of chlorinated di-phenyl in April 1930 and worked regularly until the latter part of the year 1933. About May of 1933, he noticed the appearance of blackheads on his face, neck, arms and legs. These areas itched slightly. In a short time blackheads began to appear on the chest, back and lower part of the abdomen, around the navel and on the scrotum and penis. Many of these blackheads swelled and became infected, discharging thick pus. The areas healed with difficulty and often left scars. The condition seemed to be progressive until November 1933. When seen in December 1933 the patient complained of lassitude, loss of appetite and loss of libido and said that his cutaneous condition seemed to be improving.

*Physical Examination.*—On examination he seemed in good general health. His complaint of lassitude was not borne out by anything more than the usual temperament of the Negro toward work. On the forehead, extending within the hair line, and on the cheeks, chin, nose and neck were numerous small, very black, tenacious comedones, their distribution best described as being "peppered" within the skin. Many of the comedones surmounted firm shotlike cysts, which in some areas contained viscid yellow pus. The pustular elements were more noticeable on the neck. Similar shotlike comedones and cysts had appeared on the shoulders, midportion of the back and chest, with an occasional large cyst. A peculiar peppering of the skin with tenacious carbon-colored comedones was apparent around the umbilicus and lower portion of the abdomen. The scrotum and penis were involved in a similar process, the former being given more to the formation of cysts. The outer surfaces of the forearms and anterior thighs showed similar but fewer comedones. The whole eruption was acneform but differed from acne particularly in the lack of a seborrheic appearance of the skin and in the peculiarly deep black of the comedones as well as the general peppered distribution in areas not usually involved in acne vulgaris. A general physical examination had revealed nothing of importance.

*Treatment.*—He was instructed to scrub his skin thoroughly before and after his hours of work, to wear fresh clothing each day during work and to use a lotion made up as follows: zinc sulfate 4 Gm., potassium sulfurata 4 Gm. and distilled water to make 125 cc. On recommendation he later reported for weekly doses of roentgen radiation according to the usual manner of treating acne vulgaris.

*Microscopic Examination.*—A portion of the skin of the chest was removed for microscopic study (fig. 1). The chief feature of the sections was noted in the hair follicles and sebaceous glands, in which there were cystic dilatation,

*Summary of the Symptoms and Treatment in Sixteen Cases of Acneform Eruption*

| Case | Age | Race | Type of Skin | Type of Eruption | Time of Exposure | Special Treatment |
|---|---|---|---|---|---|---|
| 1H | 22 | White | Seborrheic; previous acne | Diffuse comedones; few cysts | 6 months | Incision; roentgeno-therapy |
| 2B | 28 | White | Average | Diffuse comedones; few cysts | 5 months | None |
| 3S | 32 | White | Seborrheic | Diffuse comedones; large cysts and pustules | Throughout | Incision; drainage |
| 4W | 36 | White | Average dry | Diffuse comedones; large cysts and abscesses | 10 months | None |
| 5F | 28 | White | Average | Diffuse comedones; few cysts on face and neck | Throughout | None |
| 6S | 30 | White | Seborrheic; previous acne | Diffuse comedones; deep abscesses on neck; severe cysts | 10 months | Incision; drainage |
| 7B | 20 | Negro | Seborrheic | Erythematous diffuse comedones; few small cysts | 8 months | None |
| 8B | 19 | Negro | Average | Few scattered comedones; occasional cyst | 5 months | None |
| 9D* | 26 | Negro | Seborrheic | Diffuse comedones; cysts; small abscesses | Throughout | Roentgeno-therapy |
| 10C | 37 | White | Average | Scattered comedones; occasional abscess | 9 months | None |
| 11G | 56 | White | Dry | Scattered comedones; occasional abscess | Throughout | Roentgeno-therapy |
| 12P | 20 | White | Seborrheic | Few comedones; occasional cyst | 2 months | None |
| 13H | 37 | White | Average | Occasional comedone | Throughout | None |
| 14B | 23 | Negro | Average | Very few comedones | 12 months | None |
| 15F | 22 | White | Seborrheic | Scattered comedones | Throughout | Roentgeno-therapy |
| 16P | 20 | Negro | Seborrheic | Diffuse comedones; few cysts back and face | ? | Roentgeno-therapy |

* This case is reported in detail.

destruction of the hair, marked thinning and atrophy of the epithelium of the follicles and a heavy plug of keratinized material which partly filled the cystic cavity. In some areas there was a superficial plug at the surface opening; others showed the surface open and the plug deeply situated. There was no purulent exudate. There were a zone of moderately dense connective tissue surrounding the enlarged follicles, slight edema and infiltration by lymphocytes but no leukocytes. Slight edematous changes were noted in the occasional sebaceous glands present, but they were strikingly few. The sweat glands were normal.

Fig. 1.—Section of skin from the chest, showing histologic changes in the formation of an acneform eruption.

Of the twenty-four men working in the manufacture of chlorinated di-phenyl within the period from the late summer of 1932 to October, 1933, twenty-three were reported to have had an acneform eruption on the face and body. Of the twenty-three, sixteen were examined. These men presented eruptions of acneform character similar in type and distribution to that in the case referred to but varying in severity (table). In many patients numerous small sebaceous abscesses developed, particularly around the collora line, which exuded heavy, tenacious pus,

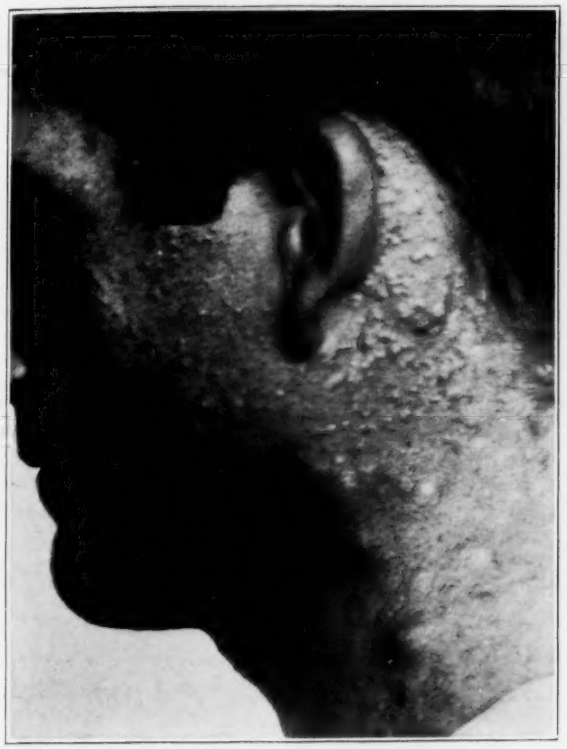

Fig. 2.—Characteristic appearance and location of an acneform eruption.

and the remaining ulcers were indolent, leaving in their wake much scarring. In two of the cases particularly, many large abscesses developed on the neck and back.

Until very recent times di-phenyl and chlorinated di-phenyl were only laboratory curiosities, and their commercial manufacture was unknown. In the experimental stage of manufacture the apparatus was necessarily crude, and experimenters and workers were exposed for long periods to fumes and dusts containing much chlorine, both free and combined. In the early experiments, as well as in the early manufacture in large

quantities, the di-phenyl was made by heating benzene derived from crude coal tar. When heated in a suitable medium benzene ($C_6H_6$) becomes di-phenyl ($C_{12}H_{10}$). This substance is then chlorinated at various saturation points, by exposing it under suitable conditions to free chlorine gas. Distillation of the resulting chlorinated di-phenyl[10] results in a purified commercial product (fig. 3). The commercial benzene always has a small quantity of impurities, but some of the cruder commercial benzenes contain relatively large quantities of these impurities, such as xylene, toluene and paraffin. On the heating of the

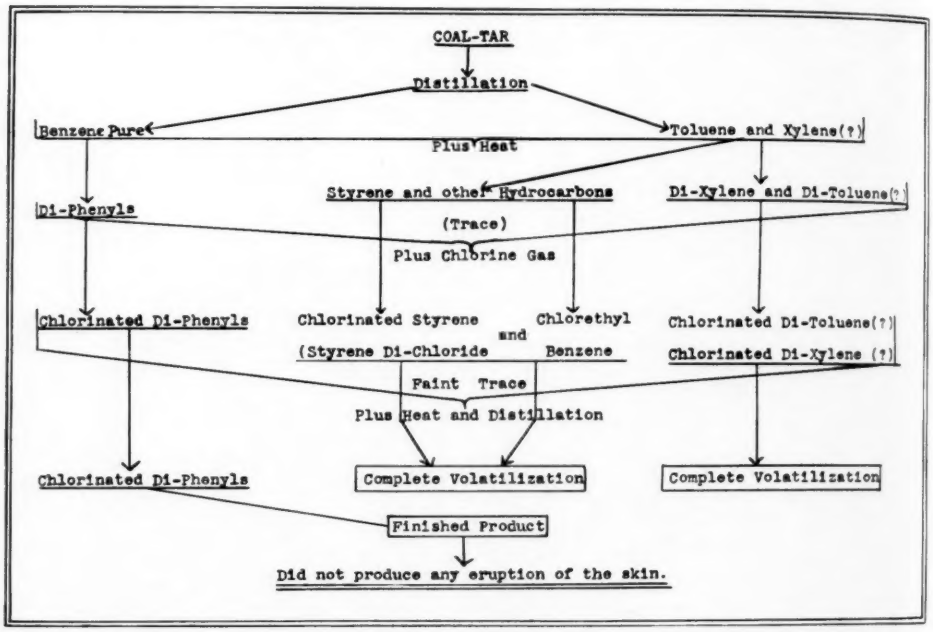


Fig. 3.—Chart illustrating the process of manufacture of chlorinated di-phenyl.

cruder benzenes, the toluenes and xylenes, when present in large quantities, may produce the chemical substance styrene, which has the formula $C_6 H_5 CH : CH_2$. Styrene when chlorinated (it being inseparable from the di-phenyl) is an unstable compound dropping its chlorine atoms very readily. It is probable that other complex hydrocarbons may be formed from these impurities, which on chlorination become quite as unstable as the styrene. One of these compounds may be chlorethyl benzene ($C_6 H_5 CH\text{-}Cl\text{-}CH_3$).

10. Chlorinated di-phenyl consists of a mixture of di-phenyl chlorinated at various saturation points: nona-chloro-di-phenyl ($C_{10}$ $HCL_4$ -$C_6$ $Cl_5$) etc.

In the experimental stage and in the early manufacture of chlorinated di-phenyl, the men working were exposed for long periods to these chlorinated products. As the demand for the finished product increased, quantitative manufacture was speeded up rapidly, and open stills and heating units were of necessity used until better equipment could be designed and made. But since no physical trouble had developed in the workers previously there was no apparent hazard connected with manufacture. On or about March 1933 the electrical properties of the chlorinated di-phenyl produced in the plant the workmen of which were examined fell below the specifications, and the color of the product deepened. About one month later an acneform eruption was observed on the faces and arms of several of the workers, which became progressively worse, gradually becoming apparent among all the workers engaged in the process at that time. Although one of the men had had a slight acneform eruption on his face in January, little thought was given to it until the eruption appeared in the others. In the summer of 1932, six or eight months previous to the general outbreak of the condition, owing to a difference in price, the crude benzene was purchased from another source, and it was not until the purchase of this particular benzene was discontinued, in October 1933, that the finished product came up to the standard. During the period from March 1933 until October 1933, the eruption continued among the men, and it was present as already described when the men were examined in December of the same year.

The appearance of the eruption coincidentally with the production of a poor grade of chlorinated di-phenyl seemed to indicate a relationship. On due chemical investigation styrene was assumed to be the offending substance causing the low grade of the finished product.

It seems that it may be readily assumed that our problem as to the causative agents of the acneform eruption was connected with the chlorinated products of the impurities present in the crude benzene. However, with the exception of styrene, these products are not known. The problem hence becomes difficult of exact solution, and it is necessary to resort to a reasonable deductive hypothesis.

The exact conditions under which the men contracted their eruption cannot be satisfactorily duplicated experimentally since prolonged mild contact seems to be a deciding factor. Repeated patch tests made on numerous persons with the finished product, chlorinated di-phenyl, and the foreign substances styrene, styrene di-chloride and chlorethylbenzene, have not resulted in an acneform eruption except in one instance. Repeated patch tests made with chlorethylbenzene in the same area produced on the third attempt an erythematous follicular eruption, which persisted only a few days but which was by no means typical.

We could not duplicate this in other instances. Since styrene, chlorinated styrene, chlorethylbenzene, xylene, toluene and their probable products are for the most part lipid solvents, they will produce death and denudation of the epithelium, and one always obtains a chemical burn in response to a patch test made with the styrene and styrene di-chloride. Small quantities mixed loosely with an inert powder or dissolved in ether produce a mild erythematous reaction but no acneform eruption. Patch tests made with the finished product, chlorinated di-phenyl, at no time produced any erythema or evidence of irritation; hence the chlorinated di-phenyl can be absolutely absolved as an irritating agent. In looking over the consecutive history of the development of the eruption, we found it apparent that the men were in some degree exposed to the chlorinated impurities in the benzene for some time before the eruption actually appeared, and that to duplicate the conditions we should have to make repeated patch tests daily for long periods in the same person, a procedure which is not feasible.

As a group, the workers in the dusts and vapors, which were presumably saturated with chlorinated hydrocarbons, were not cleanly, making little effort to bathe after work, and sweating and rubbing enhanced the liability to deposition of the substances on the skin. Theoretically, the chlorinated hydrocarbons, presumably styrene di-chloride and chlorethylbenzene, deposited on the skin gather around the hair follicles and sebaceous glands and are gradually rubbed in. Being lipid solvents, they dissolve sebum and in the reaction, which is modified by the heat of the body and the water present, give off hydrochloric acid, which causes death or irritation of the cells. This in turn causes an excess of cell growth, inflammation, sebaceous plugging and an acneform eruption (fig. 4). This process repeated over a long period may result in severe sebaceous infection, sebaceous abscess and scarring. One would naturally expect this process to occur in the seborrheic areas as well as where clothing might rub the particles into the skin. Also one would expect the process to enhance an already existing acne vulgaris. This occurred in cases 1 and 6 (table 1), the cases in which there was the worst involvement with sebaceous abscesses.

In the beginning an attempt at prevention of the condition was made by being especially careful that all men engaged in the manufacture of chlorinated di-phenyl should have a thorough bath after working hours and that they should wear freshly laundered clothing before starting work. They were also instructed to apply night and morning veterinary white lotion to the affected parts. Some of the patients having the most numerous cysts and follicular infections were instructed to report for roentgen treatment administered in the manner usually employed in treating acne vulgaris. Following the

change in the type of benzene used and the employment of enclosed distilling apparatus and ventilation fans, there was noted a gradual improvement in the acneform eruption. Those patients receiving roentgen radiation improved much more rapidly, but it cannot be said that this form of treatment gave unusual results. The final treatment, of course, was removal of the patient from the offending chlorinated hydrocarbons, and then cleanliness and relief from any infection. All the men showing evidence of cutaneous disease, so far as is known, are greatly improved, or the condition has entirely cleared except for an occasional comedo and a few scars.

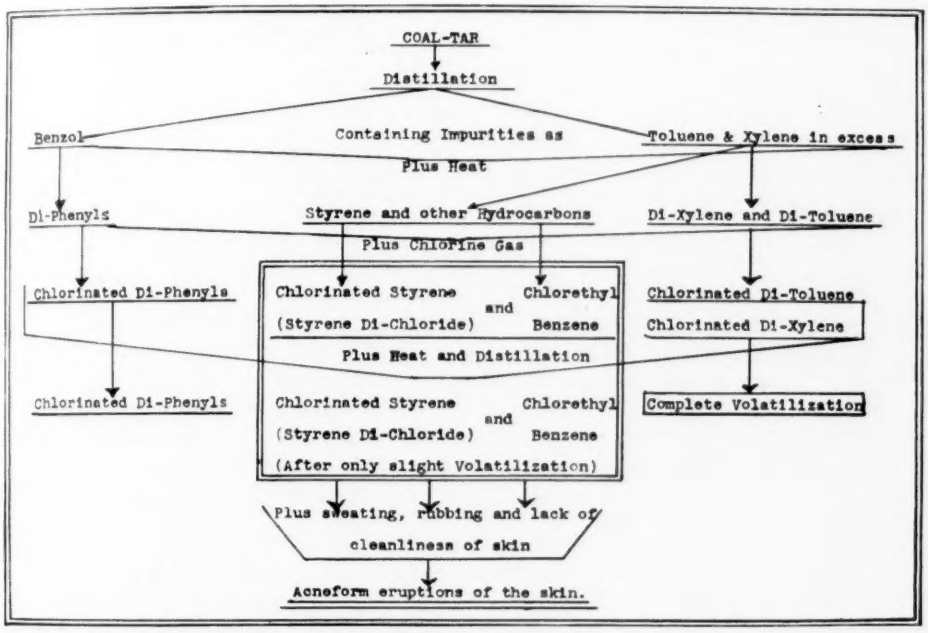


Fig. 4.—Process of manufacture of chlorinated di-phenyl containing impurities which cause an acneform eruption.

When one recalls the cases reported by the European investigators, it is altogether plausible that the chemical process outlined here occurred in most instances. The slow liberation of chlorine in the presence of a lipid solvent (usually chlorinated hydrocarbons) was present in most cases. It is doubtful whether the disease is influenced by the quantity of chlorine present; it is apparently more due to the formation of unstable chlorinated hydrocarbons as well as to the liability of the workers to close contact without a thorough cleansing of the skin and a change from saturated clothing. It is also probable that the disease has little to do with tar itself but more with the products formed by the

chlorination of tar. Hence, the disease cannot be called "chlor-acne" or "tar acne." That it is not caused by internal absorption is evident when one considers the following facts: First, acute intoxications by chlorine rarely occur, since suffocation and death by pulmonary edema ensue even in the presence of relatively small quantities of chlorine gas in the air. Second, when ingested, chlorine and the chlorinated hydrocarbons are so rapidly oxidized and chlorine so readily combines with sodium that opportunity for acne-like eruptions, such as occur in bromine intoxication, is not possible.

In retrospect, we are intrigued by the hypothesis that these theoretical considerations may have some relation to acne vulgaris. The past quarter of a century has seen an enormous increase in the use of coal for heating homes as well as in industry. The smoke stacks of the cities have belched an ever increasing stream of coal smoke—smoke heavily laden with carbonaceous products containing numerous hydrocarbons. It is well known that coal smoke contains hydrochloric acid. Is it not possible that at least some of the acne vulgaris may have a part of its origin in the constant contacts with the chlorinated hydrocarbons of coal smoke?

## SUMMARY

We have recorded herein observations concerning an industrial dermatosis of an acneform type. The type of eruption and the causative factors are analogous to those in the previously reported cases spoken of in the European literature as instances of "chlor-acne," tar acne or *Pernakrankheit*. Although exact duplication of the conditions under which the workers acquired the dermatosis cannot be accomplished by experimental means, reasonable deductions as to the cause of the difficulty can be made. The eruption occurred during the manufacture of chlorinated di-phenyl from benzene. The dermatosis occurred at a time when the benzene used contained excessive quantities of toluene, xylene and paraffin as impurities. The heating and chlorination of these impurities probably resulted in the production of styrene di-chloride and chlorethylbenzene, which, we believe, on contact with the skin produced the acneform eruption by the slow liberation of hydrochloric acid and not by internal absorption of any chemical. While many chlorinated hydrocarbons, as brought out by Prosser White,[1] are lipid substances producing death or irritation of the cells by the liberation of chlorine, this is not true in all instances, since the finished product, chlorinated di-phenyl, did not, either experimentally or actually, produce any cutaneous or sebaceous irritation. Irritation apparently depends on the ease with which chlorine atoms are released from the hydrocarbon base. We believe that these dermatoses should be labeled acneform dermatergoses resulting from certain unstable chlorinated

hydrocarbons and should not be spoken of as "chlor-acne" or tar acne. The suggestion has been made that some of the acne vulgaris of the skin may be the result of the same type of irritation from soot or coal smoke.

## ABSTRACT OF DISCUSSION

Dr. Oliver S. Ormsby, Chicago: I should like to add an experience I had three months ago that is of interest in this connection. I think that Dr. Jones' paper is extraordinarily interesting and valuable, and the small group of cases I am going to report may add to his troubles rather than solve any of the problems.

About three months ago in Michigan, I saw a patient, through the courtesy of Dr. Milton G. Butler, who presented an extraordinary acneform eruption. This patient was 18 years old and had been observed by Dr. Butler. The eruption in these various cases consisted of acneform pustules and comedones of variable size and depth, with scars, some of which were keloidal. The eruption occurred on the face and neck, particularly over the posterior portion, and extended well up into the scalp. The trunk, arms, thighs and legs were also involved. Two of the patients presented on the back of the neck deep perifollicular pustular lesions, with keloidal scars, such as are seen in Kaposi's dermatitis papillaris capillitii. Both deep and superficial atrophic scars, such as are seen in deep lesions leaving acne, were present in other portions of the neck, face and trunk. The comedones were prominent in all areas, including the legs. They were of variable size, and many were large and deep. The age of the affected patients varied from 18 to 50 years, the younger ones being more severely affected. The condition was apparently produced by contact with an ingredient in a fungicide produced by a chemical company for destroying fungi in lumber. The particular chemical in this fungicide that apparently was responsible for the eruption was sodium tetrachlor-ortho-phenylphenate, which is a yellow powder. Only those who came in contact with it had the eruption. Those who handled the other ingredients of the fungicide and did not come in contact with this particular chemical had no difficulty. As there were apparently no chlorine, bromine or tar radicals in the formula, it is difficult to determine the active factor in the production of the lesions and also whether the eruption was caused by local action by contact or through absorption and internal effects. The eruption was not concerned in any way with acne, and no patient in whom the eruption developed had had acne previously.

Dr. Marion B. Sulzberger, New York: I think that Dr. Jones has contributed a study that will be very interesting when continued. I had an opportunity of seeing a group of patients with acne of external origin about a year and a half ago. The patients were all employed in a factory making radio condensers. Pitch tar and a type of wax (halowax, a chlorinated naphthalene) were used in these condensers. The acne which developed was like that described by Dr. Jones and also included comedones on the legs, as did that which Dr. Ormsby has mentioned. I saw six patients from this one small factory. They were all in the age of puberty or the early twenties, and the group included persons of both sexes.

I believe the most important point in this study is the possible connection between acneform dermatoses distinctly due to external irritants and true acne vulgaris. It is interesting that many derivatives of tar are closely allied to the estrogenic hormone, that certain components of tar are both estrogenic and acnegenic and that the estrogenic hormone must perhaps be incriminated in the production of acne vulgaris. There can be no doubt that acne is often due to stimulation or

irritation of the pilosebaceous apparatus and that hormonal as well as external influences can produce this stimulation. In the patient suffering from acne, who has, of course, come in contact with the estrogenic hormone during intra-uterine life and then has had no further contact with this hormone (or the closely related testicular hormone) until he or she begins to form his or her own hormones, one can readily conceive the pathogenesis of acne vulgaris as being based on an excessive stimulation of the hair follicles, a stimulation produced by the sudden new formation of the hormones at puberty. It is as though the follicles, after being sensitized to the hormones during fetal contact and then passing through a period of freedom from contact, react excessively to the new and massive contacts with the products of the gonads at puberty. I have discussed this subject in great detail elsewhere.

Dr. H. G. Irvine, Minneapolis: I wish to comment on one point and to compliment Dr. Jones on an excellent piece of work and his presentation of it. There was a difference between his situation and that which Dr. Tunnacliff and I encountered (Arch. Dermat. & Syph. **33**:306 [Feb] 1936). In his case the manufacturers called on the medical profession to help solve the problem. Our study was undertaken where lay people were in charge. It shows clearly the need of medical advice in the production of various chemical preparations.

Dr. Jack W. Jones, Atlanta, Ga.: I am very grateful for the discussion. I did not have time to go into the various preventive measures attempted in controlling the outbreak or the treatment. In most of the cases the condition has finally cleared up.

Regarding Dr. Sulzberger's discussion, we studied this outbreak and the literature rather carefully for a year and a half or two years, and we are convinced that the etiology in this particular group can be ascribed to external irritation and not to any internal complications. Our idea is that the unstable chlorinated hydrocarbons give off chlorine very easily, whereas the chlorine that is fixed in the benzene ring is comparatively stable. When these unstable chlorinated hydrocarbons come into contact with the skin and enter the hair follicles, they are broken down slowly, giving off chlorine to form hydrochloric acid. We feel that this is the primary cause of the trouble.

# ACNE AND THE CARBOHYDRATES

PRELIMINARY REPORT

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There has long been an opinion among members of the medical profession that patients with acne vulgaris are intolerant to carbohydrates in general. Although this belief may still be held by many, there has been considerable evidence brought forward in the past decade which tends to refute such a belief.

A brief summary of the literature covering the evolution of this question from the dermatologic point of view is in order. Schwartz and his associates [1] reported in 1916 a series of 30 cases of acne in 50 per cent of which he found marked or borderline hyperglycemia. In 1922 Levin and Kahn [2] reported the results of an extended biochemical study on acne, in which they discovered no significant changes in the urea, nonprotein nitrogen, creatinine or calcium content of the blood or the dextrose tolerance. Twenty-nine per cent of 34 patients showed a value for the blood sugar content of 120 mg. or more per hundred cubic centimeters, however, and 30 per cent had mild acidosis. Twenty-three patients were more thoroughly investigated; 4 of these had a decreased renal threshold for dextrose, and 19 showed fermentation of carbohydrates in the feces. At about the same time McGlasson [3] maintained that acne is a condition in which hyperglycemia is an almost constant factor.

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From the Department of Dermatology and Syphilology of the Massachusetts General Hospital, E. Lawrence Oliver, M.D., Chief.

1. Schwartz, Hans J., and others: The Sugar Content of the Blood in Various Diseases of the Skin, J. Cutan. Dis. **34**:159 (March) 1916.

2. Levin, O. L., and Kahn, Max: Biochemical Studies in Disease of the Skin: II. Acne Vulgaris, Am. J. M. Sc. **164**:379 (Sept.) 1922.

3. McGlasson, I. L.: Hyperglycemia as an Etiologic Factor in Certain Dermatoses, Arch. Dermat. & Syph. **8**:665 (Nov.) 1923.

A few years later, however, dissenting comments began to appear in rapid succession. Fisher [4] was unable to confirm the previous reports; he found hyperglycemia in but 3 of 70 patients with acne. Strickler and Saylor,[5] not satisfied with single determinations, computed the values for blood sugar in eighty-five specimens for 39 patients, at varying intervals. They found cyclic variations, with but 10 per cent of the patients showing hyperglycemia. They stated: "A relationship may exist between the concentration of sugar in the blood and in the skin" and "This altered concentration of sugar might explain the great number of pyogenic lesions seen in certain types of acne." Pillsbury,[6] in studying the carbohydrate metabolism of the skin, found this organ to be a temporary storehouse for large amounts of dextrose and discovered that there are diastatic and glycolytic enzymes present in the skin. The work of these investigators also indicated that there is a fairly constant ratio between the dextrose content of the skin and that of the blood in man, although this may be disturbed following the ingestion of large amounts of sugar. A study from the angle of dextrose tolerance was carried out by Greenbaum,[7] who found too small a difference between his controls and his patients with acne to allow him to draw conclusions. He stated that if an intolerance for dextrose is associated with acne, it is not present in all types or even in all pustular types of this condition. This led him to believe that tests for dextrose tolerance are of no value, even if a connection exists between the intake of sugar and pustular acne or if intolerance for dextrose is part of an endocrine influence generally believed to be present in cases of acne vulgaris. McClendon [8] conducted studies indicating marked retention of water following the ingestion of dextrose in adults, and he expressed the belief that the skin participates in this retention of water. Rost [9] found no abnormal curves for the blood sugar content in cases of acne vulgaris, seborrhea or furunculosis. He felt that it is doubtful whether certain groups of cutaneous diseases are regularly connected with hyperglycemia and that therefore no disorder of carbohydrate metabolism was to be expected. In line with the work done by Strickler and

4. Fisher, J. E.: The Blood Sugar Level in Some of the Common Skin Disorders, Arch. Dermat. & Syph. **18**:732 (Nov.) 1928.

5. Strickler, Albert, and Saylor, M. A.: Sugar Metabolism in Acne Vulgaris, Arch. Dermat. & Syph. **20**:705 (Nov.) 1929.

6. Pillsbury, D. M.: The Intrinsic Carbohydrate Metabolism of the Skin, J. A. M. A. **96**:426 (Feb. 7) 1931.

7. Greenbaum, S. S.: Tolerance for Dextrose in Acne Vulgaris, Arch. Dermat. & Syph. **23**:1064 (June) 1931.

8. McClendon, J. F.: On the Relation of Blood Sugar to Blood Volume, and Carbohydrate to Water Retention, Am. J. Physiol. **98**:216 (Sept.) 1931.

9. Rost, G. A.: Hyperglycaemia and Skin-Diseases, Brit. J. Dermat. **44**:57 (Feb.) 1932.

Saylor.[8] Strickler and Adams[10] made 202 estimations of the blood sugar content in 101 cases of acne and found but 10 per cent of the values to be over 110 mg. per hundred cubic centimeters. In their opinion this was not more than might be expected in normal persons, so that hyperglycemia could not be said to be characteristic of acne vulgaris. Changes in the values for the blood sugar content in individual cases over periods of three months did not coincide with clinical improvement or relapse. The suggestion followed that it is possible for carbohydrates to exert an indirect effect which is not demonstrable by the present types of chemical studies of the blood. In summing up these reports the statement made by Wise and Sulzberger[11] in 1933 seems apropos: "While clinical experience tends to show that a diet high in carbohydrates and sweets seems to make some acne cases worse, there is not an iota of scientific evidence that there is any disturbance of carbohydrate metabolism in acne."

An interesting study from the standpoint of hyperglycemia and infection is that of Tauber.[12] It was based on estimations of the blood sugar content in about 1,500 cases; in one half of these dextrose tolerance tests were made. Of 514 persons with diabetes whose blood sugar contents averaged 197.4 mg. per hundred cubic centimeters, 30, or 6.1 per cent, had furuncles, carbuncles or infections of the hands or feet. The same conditions occurred in 16.2 per cent of 504 patients who received medical or surgical treatment for miscellaneous conditions, whose blood sugar contents had averaged 111.4 mg. per hundred cubic centimeters. Two and one-half times more cutaneous infections occurred in this group although the values for the blood sugar content averaged 86 mg. lower. The remaining 511 cases were all cases of dermatologic conditions. The average value for the blood sugar content in 254 patients with miscellaneous cutaneous diseases was 102.4 mg. per hundred cubic centimeters, whereas an average value of but 97.6 mg. per hundred cubic centimeters was obtained in 257 cases of furunculosis, ecthyma and generalized pyoderma. These findings led Tauber to treat 189 patients with furunculosis with a high carbohydrate diet, intravenous injections of dextrose and liver extract. His results were so striking that he expressed the belief that this therapy is almost a specific cure. The inference drawn was that the antibodies necessary for resistance to invasion by pus-forming organisms are provided by

10. Strickler, Albert, and Adams, P. D.: Blood Sugar Metabolism in Certain Dermatoses, with Special Reference to Acne Vulgaris, Arch. Dermat. & Syph. **26**:1 (July) 1932.

11. Wise, Fred, and Sulzberger, M. B.: The 1933 Year Book of Dermatology and Syphilology, Chicago, The Year Book Publishers, Inc., 1933, p. 10.

12. Tauber, E. B.: Hyperglycemia in Diseases of the Skin, Arch. Dermat. & Syph. **27**:198 (Feb.) 1933.

the dextrose and the increased intake of starch. It was this piece of work which prompted the present study. It does not seem unreasonable that if furuncles were favorably influenced by such a regimen, perhaps the more pronounced and predominantly pustular types of acne would also be aided.

## REPORT OF STUDY

It was determined that in order to carry out a carefully controlled treatment with diet and intravenous injections of dextrose the patients would of necessity have to enter the hospital rather than be handled as outpatients. Patients in whom acne was severe were not difficult to obtain. A free bed was offered, and each patient was hospitalized for a period of two weeks, during which all local therapy was withheld. A complete medical examination was made of each patient, and the usual laboratory procedures were carried out. A dextrose tolerance test was made on each patient on the morning after admission and before treatment was begun. In the ward a diet was prescribed on the following basis: protein, 1 Gm.; fat, 1 Gm., and carbohydrates, 5 Gm., per kilogram of body weight. A daily intravenous injection of 1,000 cc. of a 5 per cent solution of dextrose in physiologic solution of sodium chloride was administered. Samples of urine taken during the period when injections were being given were checked for sugar; none was found in any specimen. A test of the blood sugar content after fasting was again made on the morning of dismissal. Photographs were taken on admission and again on discharge to serve as a balance against possible prejudice in the clinical estimation of the results.

The series consisted of 10 patients, varying in age from 16 to 27 years. The duration of the acne ranged from six months to ten years; in all the cases the disease was severe and predominantly pustular. Protocols of each case would shed no more light on the situation and will be omitted. In one patient the condition suggested an element of rosacea. Another patient showed almost complete involvement of the right side of the face alone. Previous therapy had included practically all types of treatment; one patient had received roentgen therapy, 2.2 skin units having been given over three months' time, which was completed several months before admission.

The urine was normal in all the patients, except 1, who showed the slightest possible trace of albumin in two of four specimens. The same patient exhibited pronounced secondary anemia. In all the other patients the blood counts were within the normal range. All the patients gave a negative reaction to the Hinton test. The values for the blood sugar content during fasting, at the time of admission, ranged from 78 to 92 mg. per hundred cubic centimeters and averaged 84.4 mg. The results of all the sugar tolerance tests were within the normal range; two curves were of the flat type. The values for the blood sugar content on dismissal varied from 70 to 93 mg. per hundred cubic centimeters, with an average of 80.6 mg., or about four points lower than that obtained on admission.

The results of the work are of considerable interest. Of the 10 patients, none became worse; in 3 the condition was unchanged; 2 were questionably benefited, and in the remaining 5 the condition was definitely improved. The most striking observation was the fact that the erythema associated with the larger lesions became markedly lessened; this approached blanching during the time of injection in several patients. New lesions developed in only 2 of the 10 patients during the time of their stay on the ward; 1 of these showed a few superficial papules and the other a temporary slight increase in erythema. All the patients were dismissed on the diet already described, supplemented with the routine local therapy.

*Summary of Results* *

| Case | Patient | Age, Years | Duration of Condition | Urine | Blood Count | Dextrose Tolerance | Blood Sugar Values in Mg. per Hundred Cc. | | Clinical Result | Length of Follow-Up | Result of Follow-Up |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Admission | Dismissal | | | |
| 1 | J.R.K. | 17 | 1 yr. | Normal | Normal | Flat type of curve | 87 | 85 | Slight improvement | 14 mos. | Improvement continued until skin was clear |
| 2 | R.E.D. | 26 | 6 yrs. | Normal | Normal | Normal curve | 83 | 80 | Definite improvement | 13 mos. | Skin gradually cleared; slight premenstrual outbreaks only |
| 3 | M.A.K. | 27 | 9 mos. | Slightest possible trace of albumin | Secondary anemia | Normal curve | 92 | 93 | No change | Death from ulcerative colitis 4 mos. after dismissal | |
| 4 | E.C.M. | 26 | 10 yrs. | Normal | Normal | Normal curve | 80 | 75 | Definite improvement | 6 mos. | Condition stationary until roentgen therapy was given 2 months after dismissal |
| 5 | F.L.C. | 16 | 5 yrs. | Normal | Normal | Normal curve | 91 | 85 | Definite improvement | 8 mos. | Skin cleared rapidly; two mild outbreaks after eating tomatoes |
| 6 | C.A.T.H. | 20 | 4 yrs. | Normal | Normal | Normal curve | 86 | 81 | Definite improvement | 11 mos. | Flare-up 6 mos. after dismissal; roentgen therapy then given |
| 7 | A.R.B. | 21 | 3 yrs. | Normal | Normal | Normal curve | 88 | 80 | No change | 10 mos. | Condition stationary until roentgen therapy was given 2 mos. after dismissal |
| 8 | I.I.C. | 17 | 6 mos. | Normal | Normal | Flat type of curve | 78 | 70 | No change | 9 mos. | Skin cleared rapidly |
| 9 | J.N.L. | 20 | 18 mos. | Normal | Normal | Normal curve | 83 | 75 | Definite improvement | 8 mos. | Skin gradually cleared |
| 10 | V.P.N. | 19 | 5 yrs. | Normal | Normal | Normal curve | 80 | 82 | Slight improvement | ..... | Patient could not be located |

* As an immediate result of the treatment 50 per cent of the patients showed improvement. In the follow-up 50 per cent continued to show improvement until the skin was clear.

The follow-up of these patients has extended over periods of from six to fourteen months. One patient could not be followed. The patient who showed albuminuria and secondary anemia was later found to have ulcerative colitis and died following an ileostomy. Three patients have subsequently been treated with the roentgen rays and have responded satisfactorily; 1 did well for six months before this treatment became necessary. The remaining 5 patients have continued to improve with only the high carbohydrate diet and local therapy; they are practically free from the eruption at the present time. One of these is a woman who shows no signs except a slight premenstrual outbreak; another has been troubled by two mild flare-ups, each of which immediately followed the ingestion of tomatoes. It was interesting to note that not all the last-mentioned 5 patients had shown definite improvement during their stay in the ward.

## COMMENT

It was not expected that the high carbohydrate regimen would prove to be the ideal means of handling acne vulgaris. The results obtained in this study, however, seem to indicate not only that the old theory of a low intake of carbohydrates is inapplicable as a general measure but also that diametrically opposite treatment is often helpful.

The objection may be raised that hospitalization for a period of two weeks could of itself bring about an improvement in the average case of acne. This may be true, but the patients in this series were not bed patients in any sense of the word; they were in bed only during the time consumed by the intravenous therapy and were allowed the freedom of the ward otherwise. The fact that 50 per cent of them have continued to show improvement since dismissal also militates against the possibility that the mere stay in the hospital was a major factor.

The study of the series reported on here is but the beginning of the study as planned. A second experiment has been started in which the patients are receiving the same diet under the same conditions but in which solution of sodium chloride alone is administered intravenously. This will perhaps show how extensive a part is played by the fluid as such. As yet an insufficient number of patients have been tested to justify a report. After a study of the second series is completed other studies will be carried out, with the use of varying concentrations of the dextrose solution and also with the diet alone.

The fact that individual foods may possibly be important factors in certain cases was suggested by the report that 1 patient had two exacerbations directly after the ingestion of tomatoes. In another case there was a history of attacks which were attributed to a particular kind of home-made candy, whereas other more simple candies were innocuous. Chocolate is, of course, universally recognized as a notorious offender. Specific foods have been isolated as noxious by some observers for many years. Charles J. White,[13] as long as fifteen years

13. White, C. J.: Personal communication to the authors.

ago, stated that cooked fats are at fault. Recently, Cleveland White [14] has studied the question of specific foods in cases of acneform eruptions. He has expressed the belief that in some of these cases the condition is based on a definite sensitization. He reported on a series of 34 patients in whom causative food allergens were determined by the use of elimination or "nonallergic" trial diets, as ordinary cutaneous tests were of little or no value. The foods most frequently found to be offenders in his cases were chocolate, milk, wheat, oranges, tomatoes and nuts. The question may arise here whether such foods may be an indirect cause of a digestive disturbance rather than primary factors in bringing about the acneform process.

## SUMMARY AND CONCLUSIONS

A short résumé of the literature indicates that the trend of belief is away from incriminating faulty carbohydrate metabolism as a factor in the mechanism of acne vulgaris and of furunculosis. As the first step in a study of this question, a series of 10 patients with severe pustular acne were hospitalized and given routine examinations, including the usual laboratory studies and dextrose tolerance tests, and the blood sugar content was again checked on dismissal. All the patients showed values for the blood sugar content in the lower range of normal at all times and gave normal reactions to dextrose tolerance tests. They were placed on a high intake of carbohydrates and were given daily intravenous injections of dextrose for two weeks. Fifty per cent of the patients exhibited definite improvement; 20 per cent showed a slight improvement, and none was worse. Maintenance of this diet over periods up to one year has resulted in continued improvement likewise in 50 per cent of the patients, with routine local measures as the only adjuvant. The results of this experiment definitely intimate that a high carbohydrate regimen is not inimical to the welfare of patients with acne. It is possible that other types of foods or, perhaps, specific foods, are more to be incriminated as factors in cases of acne than the long abused carbohydrates.

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14. White, Cleveland: Food Sensitization Dermatoses: Preliminary Report, J. Allergy **4**:151 (Jan.) 1933; Acneform Eruptions of the Face: Etiologic Importance of Specific Foods, J. A. M. A. **103**:1277 (Oct. 27) 1934.

# PARADOXICAL INFLUENCE OF LIGHT RAYS AS A CAUSATIVE AND AS A CURATIVE FACTOR IN CANCER OF THE SKIN

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The title of my paper requires elucidation from two standpoints. First, it is not my intent to take a definite position as to the ultimate etiologic factor in the production of cancer of the skin. Rather, I shall attempt to show to what extent radiant energy is able to produce a predisposition to the development of malignant degeneration and to demonstrate the mechanism of its activity in this direction. It is apart from my purpose to prove that the cellular damage caused by various rays is able alone to cause cancer, as it is obvious that one may be dealing merely with a soil made favorable for the development of cancer rather than with rays as an immediate causative agent.

It shall be my purpose to attempt to explain whether the paradoxical effect of light rays in the causation of malignant degeneration and in its cure is the result of different biologic effects of the rays on the cell, or whether it is the same biologic mechanism with a different end-result.

Until very recent times, this problem seemed to have been satisfactorily settled. However, in the last fifteen years, on the one hand a number of new fundamental scientific facts have appeared which have bearing on this question, and on the other hand there is an entirely new interpretation as well as regrouping of previously established facts. During the past ten years the entire theoretical structure of the biologic action of light rays has been discussed, and the conclusions have led to practical improvement in the treatment of cancer of the skin by radiant energy.

At the threshold of the discussion, one must take the stand that there is no difference in the biologic action of the various light rays. However, in the curative sense, only roentgen rays and radioactive substances come into the problem, while as causative factors, not only these, but the ultraviolet rays are of very great importance. Time does not permit

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Studies and Contributions from the Department of Dermatology and Syphilology, University of Michigan Medical School, service of Dr. Udo J. Wile.

me to go into minute detail as to the physical mechanism of the action of radiant energy. I shall state only that it is conceded that electromagnetic oscillations sufficiently strong split off electrons from the cell atoms. In other words, the electrons are displaced from their course near the nucleus to the periphery of the atoms. These disturbances cause a great alteration in the cell in every respect. The action of the so-called corpuscular or beta and alpha rays is in its final result identical with that of electromagnetic vibrations. Impinging on the tissues, these rays set up electromagnetic vibrations, which, in turn, reproduce beta and alpha rays.

I should like to emphasize, at this point, that the visible and caloric rays do not come into consideration in the problem under discussion. The reason for this is that the quanta of these waves are not large enough to split off electrons from the atoms and to displace them to the peripheral courses of the atoms.

The size of the quantum of the caloric waves, taken as a unit, establishes the ultraviolet quantum as two or three times higher. Depending on the wavelength, the quantum of roentgen rays is from 720 to 70,000 times higher than the caloric unit, and that of gamma rays, 720,000 times higher.

The quantum of the caloric rays, therefore, is not sufficiently strong to exercise a substantial effect on the atom. The caloric rays, therefore, may be considered only as a physical remedy causing destruction by actual heat. In this sense, they are unimportant either as immediate or remote factors in the causation of cancer of the skin. When, under unusual circumstances, cancer occurs following a burn or a therapeutic burning procedure, it is analogous to that occurring in scar tissue and in no way is associated with the cancer induced by radiant energy. Cancer never occurs following such therapeutic procedures as diathermy, unless actual burning has taken place. The same thing is true of the so-called kangri cancer, which starts from a scar as a result of a burn and is not analogous to cancer occurring after physiologic and pathologic irradiation.

I am concerned, therefore, only with ultraviolet rays, roentgen rays and the rays of radioactive substances. These all work in the same manner, differing only in the strength of the effect produced. The differences of these effects are determined, first, by the size of the quantum and, second, by the difference in the absorption of the rays by the tissue affected.

These facts established, I must discuss the changes produced in the tissue by such radiation. Two questions present themselves for elucidation: First, do all tissues react in a similar way to radiation of the same strength, or is there a qualitative difference in their reaction?

Second, do differences in the strength of the same radiant energy exercise the same or different effects on the same tissue?

With regard to the first question there is unanimity that in any tissue the biologic effect will vary only in degree. It is only in this variance in degree that one can speak of an elective biologic effect on tissue.

With regard to the second question there is still wide divergence of opinion.

According to the so-called law of Arndt and Schulz, a very small dose of radiation exerts a biologic action different from that of a large one. There are many who still support this view.

The interpretation of Arndt and Schulz bears directly on the salient point of the paper in relation to the causative versus the curative effect of radiation. Small doses of radiant energy distributed over a long period would in this sense act as a causative factor, while the large doses required in the treatment of cancer of the skin would act as a curative agent. If, therefore, the Arndt and Schulz theory is true for the biologic effect of radiation, there is a true paradox in the action of light rays in both curing and causing cancer and not a paradoxical end-result alone.

The explanation of Arndt and Schulz presupposes that small doses stimulate functional activity of the cell, whereas the higher doses paralyze these functions and still higher ones result in cell death.

There is considerable confusion when one reads the German literature as to the interpretation of the word *Reiz*. This word implies both stimulation and irritation. There is no question that irradiation spells irritation for the cells. The real question for discussion is whether a small dose of radiation can cause stimulation in the sense of increased functional activity. Before carrying this discussion further, it is proper to note that the law of Arndt and Schulz, stated first on pharmacologic grounds, is disputed by such eminent authorities in pharmacology as Meyer and Heubner, and the roentgenologic accuracy of this observation is especially questioned by such authorities as Holzknecht and Pordes.

The literature of the last ten years on the subject seems to favor the views of Holzknecht and Pordes [1] that, so far as roentgen rays and gamma rays are concerned, stimulation of cells does not take place. It is more difficult to take a positive view with regard to the effect of ultraviolet rays, since constant irradiation with these occurs in the natural environment, and it is therefore difficult to determine what constitutes an additional dose.

1. Holzknecht and Pordes: Strahlentherapie **20**:555, 1925.

It is known that the lack of such rays causes underdevelopment. Thus, certain salamanders living in the darkness of caves are not pigmented and are sightless. When, however, these animals are exposed to light, they acquire pigment and organs of vision. This can hardly be attributed to a stimulation in the pathologic sense but merely to the development under the influence of light to a normal state. In this connection, there are some interesting experiments on the increase in growth of plants under irradiation. Reiss [2] determined that following irradiation there was an increase in the development of the buds of the lilac. He found, however, that this increase was merely an increase in water content. He was not able to determine any marked increase in cellular activity or in number of cells. The experiments, however, of Schwarz,[3] Guilleminot [4] and others along similar lines seem to suggest that under irradiation there is increased cellular proliferation. Of great interest are the experiments of Weber,[5] who found that by irradiating lilac buds they could be made to bloom much earlier, but the plant rapidly perished. In like manner, the irradiation of frogs' eggs and the bones of young rabbits and dogs by Hoffmann [6] resulted in a much more rapid cellular development, which, however, was very temporary, as controls had caught up with the experimental material within two or three weeks. The analysis of these findings suggests in the one instance an increased cellular activity owing to irradiation which rapidly led to cell death, as in the experiments on the lilac. Other experiments seem to indicate a temporary stimulation of cellular activity, but the cessation of this activity after increased growth and a relapse to normal growth suggest cellular injury, the occurrence of which I hope to prove later.

It is incontestable that irradiation may have the effect of increasing the proliferation of the cancer. This is a common experience. Ritter and Lewandowsky,[7] for example, treated the cutaneous metastasis of ovarian cancer with various doses of roentgen radiation. They described a proliferation following the application of small doses and a disappearance after large ones. I have emphasized the importance of this in a book by Böhmer and myself,[8] "Light Treatment in Cutaneous Diseases," by stating that in the irradiation of malignant tumors it is necessary to give a dose large enough to destroy the tumor tissue. If

2. Reiss: Compt. rend. Soc. de biol. **92**:987, 1925.
3. Schwarz, G.: Arch. f. d. ges. Physiol. **100**:532, 1903.
4. Guilleminot: Encyclopédie scientifique, Paris, Doin et fils, 1910.
5. Weber, Friedl: Arch. f. d. ges. Physiol. **198**:644, 1925.
6. Hoffmann: Strahlentherapie **13**:285, 1922; **14**:516, 1923.
7. Ritter and Lewandowsky: Strahlentherapie **4**:412, 1914.
8. Blumenthal, Franz, and Böhmer, Lothar: Strahlenbehandlung bei Hautkrankheiten, Berlin, S. Karger, 1932.

one gives less than this dose one frequently sees an increase in the growth in both width and depth, and this is so common that it cannot be regarded as a coincidence.

This phenomenon, however, is explainable on grounds other than direct stimulation of the cancer cell. Gatenby,[9] for example, following irradiation of tumors with one one-hundredth erythema dose observed cessation of mitoses in the cancer cells and appearance of abnormal mitoses. The increase in growth of a malignant tumor following underdosage can therefore be ascribed to the effect of proliferation of the remaining cancer cells caused either by the irritation and inflammation arising in the subcutaneous tissue or by the irritation exerted by the death of certain of the affected cancer cells, rather than by the stimulation of the light rays.

I do not believe that there are any experiments which prove conclusively that there is a distinct biologic difference between the primary effects of small and large doses on the individual cell. My own view is that both small and large doses inflict an initial injury. The initial injury under small doses produces changes which, in some instances, simulate cell stimulation. The effect of irradiation in relation to dosage differs, then, only in degree, the larger doses producing cell death, the smaller ones provoking cellular disturbances which in effect are inimical to the further normal existence of the cell. Here one must distinguish the cell from the organism as a whole. The effect of small doses on cellular activity of certain types—for example, that in psoriasis—is inimical to the cell but beneficial to the host, and it is in this way that the therapeutic effects of small or divided doses of roentgen radiation are explainable.

I can now enter the discussion of the causative factors in the production of cancer as they refer to light rays, on the one hand, and the curative potentialities of light rays, on the other.

In what manner does radiant energy or do rays of light affect the activity of cells and tissue? In this connection, there are some interesting experiments with protozoa and lower animal forms. These experiments concern themselves not with the immediate effect of irradiation but with its ultimate results. There is, of course, a latent period between irradiation and the first appearance of change. There has been great dissension as to the manner in which the cell is injured by irradiation. It is now definitely established, largely through the work of Hertwig[10] and of Halberstaedter,[11] that the nucleus is the chief point of injury.

9. Gatenby: Irish J. M. Sc. **48**:748, 1929.

10. Hertwig: Strahleneinwirkung auf Wachstum und Entwicklung, in Lazarus, P.: Handbuch der gesammten Strahlenheilkunde, Munich, J. F. Bergmann, 1927, vol. 1, p. 444.

11. Halberstaedter, L.: Berl. klin. Wchnschr. **51**:252, 1914.

The question at issue, however, is first whether the nucleus is directly injured by light rays, or whether it is secondarily changed by the immediate injury to the protoplasm. Working with trypanosomes, Halberstaedter determined that the first injury to the cell from irradiation is in its generative function, that is to say, its ability to divide. This was later shown in the frog egg by Hertwig. There are, of course, many other changes which occur in the cell from irradiation. For my purposes, the change in the power of regeneration is of especial interest. Hertwig and Müller, working independently, demonstrated that irradiation of both the egg and the spermatozoa of the frog produces no apparent structural change but results in a very large number of deformities. From these experiments, Hertwig concluded that the function of differentiation of a cell is disturbed if its nucleus is harmed.

It is just such differentiating cells which one finds in the basal layer of the epidermis. These cells perform all the work of differentiation as well as that of regeneration of the epidermis. In this layer and in the changes which take place following irradiation one must look for the understanding of the point under discussion, namely, the curative and causative factors due to the action of rays in relation to cancer of the skin. In connection with the causative effect, that is to say the changes that take place in the basal layer which lead directly or indirectly to cancer, it is best to consider at first the ultraviolet rays, and of these the so-called Dorno rays, or rays of 3,000 angstroms, are of the greatest importance for discussion. I omit for the present notice of the action of gamma rays and roentgen rays, and turn to the ultraviolet rays for the reason that in the application of roentgen and gamma rays there are added factors in injury to the subcutaneous tissues and blood vessels. Bachem demonstrated that 66 per cent of ultraviolet rays is absorbed in the superficial layers of the epidermis and that the basal layer and the cutis each absorb about 16 per cent. From this it is possible to see the definite protective function of the horny layer against these rays.

Ultraviolet rays in doses surpassing the physiologic limit produce characteristic changes in the basal cell layer. According to Miescher,[12] the damaged cells show all the signs of pathologic karyokinesis. After two or three days these cells can be seen in the horny layer, where their nuclei are retained. At the very outset leukocytes are seen emigrating from the cutis into the epidermis, but this mild infiltration is not sufficient to produce the changes mentioned, which are without doubt the effect of the rays acting on the nuclei of the basal cells. The greatest emphasis must be put on this point. It is known that the basal cells produce two protective measures against the rays: horny substance as a product of the nucleus and pigment as a product of protoplasm.

12. Miescher, G.: Strahlentherapie **45**:201, 1932.

Certainly it is not accident that the most impressive changes in precanceroses due to radiation are found as hyperkeratoses and hyperpigmentation, and that developing cancer finds its starting place in tissue so changed. In this fact one again meets an apparent paradox. The very reaction of the cell as a protective measure against the rays seems at the same time to be the starting point for cell degeneration.

Kyrle [13] took the view that pigmentation and hyperpigmentation are functions purely protective against the ultraviolet rays, and he further regarded keratinization and hyperkeratinization as in no way concerned with the defense mechanism against the rays. According to his view, proliferation and pigmentation are not coordinate, but proliferation follows pigmentation. He advanced the idea that those cells which have lost the power to produce pigment give all their strength to proliferation. I cannot agree with this view. It is well known that pigmentation and thickening of the horny layer are both protective mechanisms against the rays; the immediate thickening of the horny layer in fair and red-haired persons exposed to ultraviolet rays is so impressive that it overshadows the protective mechanism of the pigment. The importance, however, of the pigment is clearly indicated in the fact that the Negro's skin is ten times less sensitive to ultraviolet rays than the white person's skin. Pigmentation and keratinization are definitely coordinating functions of a mechanism protective against these rays.

If the cells of the basal layer receive a quantity of radiant energy exceeding physical limits, a third process starts, and this is a degenerative one, just such as occurs in eggs and spermatozoa following irradiation. This degeneration is not a result of increase of pigment, nor is it due to proliferation of the epidermis, but it is due to an inherent loss or lack of a protective function. Statistically, people lacking in pigmentation or more exposed to ultraviolet rays are found with the highest percentage of cancer of the skin. This is evidence in support of the fact just stated.

Statistics have shown that cancer of the skin in the majority of cases is localized in those places exposed to ultraviolet rays. Red-haired and fair persons having occupations which expose them to these rays are definitely predisposed.

I state it as my belief that the degeneration in the epidermis starts very early following irradiation and that the continued application of small doses leads ultimately to visible changes in the skin until finally malignant degeneration takes place. The developing neoplasm occurs in the places of greatest proliferation; therefore it always begins in a wartlike hyperkeratosis, which it is perfectly proper to characterize as a precancerous change.

13. Kyrle: Vorlesungen über Histobiologie der menschlichen Haut und ihrer Erkrankungen, Berlin, Julius Springer, 1925, vol. 1.

From the clinical standpoint, the changes in the skin affected by various types of rays give a very uniform picture. They differ materially only in the element of time or duration and the saturation of the insult.[14] The changes seen after a lifetime of exposure to sunlight are in no way different from those produced by a single overdose of roentgen radiation, and the reaction to the insult will differ markedly according to the race and color of the subject, but only in degree. Thus, the senescent changes due to saturation with sunlight occur in blonds much earlier than in darker persons, and they occur at an extremely early age in those patients who have a congenital defect in their resistance to light.

It remains for me now to discuss the question, In what manner does a precancerous change develop into cancer of the skin?

Bordier,[15] on the basis of a very large clinical experience, stated that a cancer develops from a precancerous lesion not only as the result of a continuation of the initial insult but as a result of any continued trauma. This fact is extremely important because the transformation from a precancerous lesion to cancer does not require the same insult which initiated the lesion. Any irritation or casual trauma can act as the determining insult. Cancer develops from arsenical keratoses in which the arsenic as the initial factor in the development of the hyperkeratosis has long ceased to exist, and the cancer usually ensues from an external traumatic source. The same thing is occasionally seen in cancer caused by roentgen radiation. The first stage is initiated as precancerosis by the rays; the final stage is initiated by friction of clothing or other casual traumatic incidents. The salient point in this discussion is that ultraviolet rays do not cause cancer in themselves. They produce characteristic degenerative and regenerative cell changes leading to precancerous lesions in the skin. With such changes in situ, any irritation, including additional ultraviolet rays, can cause the precancerous change to become malignant. It must be admitted that only a small number of precancerous changes, even those subjected to continuous trauma, undergo malignant degeneration. Expressed otherwise, all persons with precancerous changes do not necessarily acquire cancer. For this reason, one must assume another factor, which does not come within the realm of this paper for explanation, but which is of great importance, namely, a predisposition or an inherent tendency to malignant degeneration which must be based on the factor of constitution.

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14. As Dr. W. A. Pusey pointed out [Science **33**:1001 (June 30) 1911], radiant energy gives an exaggerated picture of the senile skin, and the starting point for the cancer in both conditions is situated in the keratoses. He stated the belief that the senile changes of the skin are in good part the result of the less powerful action over a long period of years of sunlight.

15. Bordier: Paris méd. **1**:109 (Feb. 4) 1933.

I now come to the mechanism of healing which follows irradiation, and here again there are two explanations offered for the disappearance of cancer cells under the influence of radiant energy. On the one hand, Opitz,[16] Theilhaber,[17] Fränkel,[18] Ricker [19] and others have expressed the belief that the disappearance of cancer cells is indirectly due to changes in the connective tissue of the stroma. They have held that the death of the cancer tissue is not a direct action of the rays but results from secondary changes affecting the connective tissue as well as the blood vessels. Some are so convinced of this that they believe it unwise to use such doses as may injure irreparably the protective mechanism of the connective tissue.

On the other hand, and directly opposing this view is that held by the majority of observers that death of the cancer cell is directly due to the action of the rays on the cancer cell itself. If this view is correct, the production of cancer by radiant energy and its cure by the same agent occur by the same mechanism, as between the degenerative and the lethal effect there is merely a difference of degree. Therefore, the same physical mechanism leads to a paradoxical end-result.

A great many clinical and histologic researches are concerned with this particular question. From the clinical standpoint, it is always the experience that cell death in cancer of the skin results only from the application of large doses of radiant energy, and that disappearance and cure never occur following small doses. This speaks for the necessity of direct action on the cancer cell to insure its death and the cure of the lesion. These clinical findings are in complete accord with the histologic pictures in cancer tissue which has been irradiated. It is an accepted fact that the most striking changes first seen occur in the nuclei of the cancer cells. The nucleus falls to pieces, karyorrhexis, karyolysis, giant nuclei and giant cell formation and vacuolation characterize the picture. One finds vacuoles, edema, lipoid degeneration, small hyaline bodies, granulation and lysis of the protoplasm. In short, the cancer cells show all phases of degeneration. Without question, the stroma is also changed but not in such degree that one could in any way explain the cell degeneration as subsequent to it or caused by it. In this connection, Perthes [20] has shown that changes in the cancer cell can be demonstrated before any change takes place in the connective tissue.

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16. Opitz: Monatschr. f. Geburtsh. u. Gynäk. **61**:232, 1923.
17. Theilhaber, A.: Strahlentherapie **11**:686, 1920.
18. Fränkel: Zentralbl. f. Gynäk. **44**:1285, 1920.
19. Ricker: Strahlentherapie **5**:679, 1915.
20. Perthes: Strahlentherapie **14**:738, 1923.

That the connective tissue changes are secondary or come as a later event following irradiation is the final conclusion of Lubarsch and Waetjen,[21] who have published an extensive monograph on the subject.

One may conclude, therefore, that the degenerative effect of radiant energy, which on the one hand leads to the development of cancer tissue and on the other to cure of the same tissue which it provokes, constitutes a true example of a paradoxical fact in medicine.

Differences in the dose and in the method of administering the rays lead to quite different end-results; small repeated doses lead to senescent changes such as farmer's skin, xeroderma pigmentosum, roentgen-ray dermatitis and radiodermatitis, all conditions concerning the precancerous nature of which there is no doubt. On the other hand, large doses lead to the cure of cancer. Small doses lead to a morbid condition of the cell and large doses to death of the cell. Still more paradoxical, it seems to me, is the fact that a massive dose intended to destroy cancer may produce cancer in the area irradiated. I cite here an experience of Dr. Wile's: A woman, following amputation of the breast for cancer, received a large dose of roentgen radiation to the axilla. She remained free from any appreciable effect for fifteen years. After this time, telangiectasis and hyperkeratoses developed rapidly and quickly led to malignant degeneration. This paradoxical end-effect may be explained on the basis which I have set forth already. It is known that in irradiation not all cells are injured to the same degree. The cells in the process of mitosis and in that immediately following it are the most sensitive. The remainder, apparently slightly or not at all injured, take part in the regenerative process. They have received, however, more or less of the degenerative injury, which is responsible for the later visible degenerative processes in the areas injured, and these later lead to cancer.

## CONCLUSIONS

1. I believe that in irradiation the mechanism acting on the cells exercises paradoxical end-effect in the development of cancer on the one hand and its cure on the other.

2. In both these processes, one is concerned with a degenerative effect in the cell particularly affecting its nucleus.

3. Dependent on the degree of the irradiation, ultraviolet rays, roentgen rays and rays of radioactive substances behave in exactly the same manner whether they lead to repair, to permanent degeneration and hence to malignant degeneration or to cell death resulting in cure of the cancer.

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21. Lubarsch and Waetjen: Allgemeine und spezielle Histologie der Strahlenwirkung, in Lazarus, P.: Handbuch der gesammten Strahlenheilkunde, Munich, J. F. Bergmann, 1927, vol. 1, p. 304.

# J. FRANCIS AITKEN

## AN APPRECIATIVE SKETCH

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This paper does not pretend to be a biography, an obituary or a eulogy. My sole reason for its presentation is my deep affection for Aitken, which has extended over many years, and I hope that I may make him seem real to those who did not know him and refresh the memories of those who knew him well.

In many respects Aitken was an extraordinary person. He was a member of no dermatologic society other than the Section of Dermatology and Syphilis of the New York Academy of Medicine, the sessions of which he attended irregularly and only as a listener. He refused all offices and did little, if any, writing; yet he was considered one of the most eminent clinicians in New York and second to none in diagnostic ability. In my opinion, based on intimate contact with him for twenty years, this seeming neglect of the higher things in dermatology was due entirely to an innate modesty and diffidence; he hated the limelight to such an extent that he could not be induced to air his opinions in public, but in his own circle of intimates he did not hesitate to venture his views, and they always proved of great value. Aitken was highly intelligent, with great reasoning ability and a marked degree of common sense. This combination of qualities, together with the personal inspection and therapeutic direction of, conservatively, some 10,000 patients a year for forty-four years, developed him into a master clinician, with a thorough knowledge of the many-faceted variations of the commoner dermatoses.

It must be difficult for the younger dermatologists to realize the diagnostic ability and therapeutic results attained by the pioneers of yesterday. These eminent dermatologists made up for their lack of laboratory facilities with unusually trained analytic and photographic minds, together with exhaustive clinical notes and an extraordinary development of what might be called sight diagnosis and what I sometimes call the sixth dermatologic sense. Jonathan Hutchinson is a good example of a physician with this peculiar gift, as is also Alfred Fournier. Their therapeutic results were surprisingly good. The old-fashioned curet, the electric needle, the cautery point, the comedo extractor and

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even an ointment accomplished results which would surprise and even astound the fledgling dermatologist of today, accustomed as he is to the innumerable paraphernalia of the modern dermatologist's office.

Of all the physicians who have served the New York Skin and Cancer Hospital, I believe that I can say, without fear of contradiction, that J. Francis Aitken gave more of himself in its service than any one else. He was house physician from Dec. 1, 1883, to Dec. 1, 1884, and thereafter became, successively, clinical assistant, assistant physician and attending physician; he held the last position until a year or so before his death on Aug. 3, 1930. Therefore, for forty-four years he attended the hospital three days a week, and during the twenty years that I knew him he never failed to be present at 2 p. m., never missed a day, other than a few weeks in the summer, always faithfully made his rounds and attended meetings of the Medical Board, and this in all kinds of weather, no matter how ill he might have felt at times. Such was his service, and in my estimation it deserves far more recognition from a hospital than the greatest of financial contributions. Such a record deserves to be emblazoned to high heaven and serve as an example to all physicians.

Aitken was born on Oct. 28, 1851, and graduated from the Bellevue Medical College on March 14, 1883. He was the first to serve as house physician at the New York Skin and Cancer Hospital. When he became the only member of its house staff the hospital had been in existence for almost a year, as it was opened on Jan. 11, 1883. The entire staff consisted of L. Duncan Bulkley and George Henry Fox as attending physicians and Daniel Lewis and W. T. Alexander as assistants. A special department for internal cancer in females was in charge of J. B. Hunter and J. D. Anway. In the first year 774 patients were treated, including 49 with cancer. This was the cradle of Aitken's dermatologic life. It was here that he developed the diagnostic acumen which we admired so much. He was probably the greatest exponent and propagandist of sight diagnosis in dermatology whom I can recall. I have frequently heard Fred Wise express the wish for the return of sight diagnosis as taught by Aitken in preference to complete dependence on laboratory reports. Aitken always made a diagnosis first; he disliked having any one question the patient until all had expressed an opinion. Such a method naturally stimulated his keenness of observation, and that was exactly what he always tried to inculcate in all his associates. He occasionally "muffed" a diagnosis, as he was the first to admit, but his accuracy, on the whole, was phenomenal. Even at times when his subordinates thought that he was wrong, time proved him eventually right. He liked to surround himself with his staff and make every one, even the youngest intern, offer a diagnosis and then patiently point out the many mistakes that we had made.

He was especially fond of the diagnosis pityriasis maculata et circinata, which, as may be surmised, is better designated as pityriasis rosea; yet in the use of the former term he had distinguished company, for both Bázin and Hardy used it in describing pityriasis rosea long after Gibert had given it its proper name. He resented most being told the latest theories by the youngest "know-it-all," which is so common in every clinic; at such times, particularly if it had been a hard and hot afternoon, he would literally explode.

After all the patients had been seen he often regaled us with tales of old New York. In his younger days he was fond of horse-racing, and he always drove a handsome pair of bays. He had a convivial nature and often spent an afternoon or evening at the old Hoffman House or some other well known hostelry with such old cronies as Stuyvesant Fish and Stanford White.

I believe that it was during Aitken's incumbency at the clinic that a woman suffering from chronic eczema misunderstood the instructions and swallowed a teaspoonful of calamine lotion three times a day for a week and at the same time generously patted on the eruption her rhubarb and soda mixture. On her return after a week of this bizarre treatment, instead of presenting alarming signs of phenol poisoning, she seemed greatly improved, whereupon an ointment containing 4 per cent rhubarb was used for many years in the clinic in the treatment of eczema.

Aitken was stout and florid. On hot days great beads of perspiration stood out on his forehead. He always was in shirt-sleeves and occasionally wore vivid-colored garters on his arms. He always carried the largest magnifying glass that I have ever seen, through which he peered at the patients and with which he would scare the wits out of them when he was especially vehement.

Aitken sought no honors. He did not even get an appointment as Attending Physician at the hospital until 1912, thereby serving twenty-eight years as an assistant, despite his great ability and faithful attendance. He invariably referred to a superintendent who took herself most seriously as "the Matron," much to her obvious disgust. Aitken always remained aloof from hospital politics, and when he did take sides it was always for justice's sake. He was guileless, and his rough exterior covered a great heart.

He practiced in the same location on East Thirty-Fifth Street for more than fifty years, and in that time he brought comfort and relief to many sufferers. I remained at his elbow for twenty years, and the proudest moments of my life were when he called me "son," which he frequently did in his later years. J. Francis Aitken lived and died fearlessly. He loved life, his specialty and his fellow-men. Need more be said other than that we who knew him best loved him most?

# CURABILITY OF SYPHILIS

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Theoretically and practically syphilis is curable. However, it is impossible to decide when the last spirochete has been eradicated in the individual patient. This question is nevertheless of grave moment to the community, to the infected person and to the physician. Under modern conditions clinical and serologic "cure" is readily obtained in most instances. Radical biologic cure with complete destruction of the last spirochete is a therapeutic ideal which probably is attained frequently when intensive and continuous therapy is used during the early stages of the disease, especially in patients with primary syphilis, whose serologic reactions are negative. At present there is no reliable test for the determination of the presence or absence of the spirochetes in the human body. The various serologic tests merely indicate a reaction on the part of the body against the spirochete. When these tests are negative there is no proof that the spirochete is not present in a state of latency. There is a tendency to relapse after any plan of antisyphilitic therapy. Stokes and his associates [1] have reported the incidence of cutaneous and mucous relapses as 9.6 per cent in patients with an early stage of syphilis who received treatment with arsphenamine and as 3.6 per cent after the use of arsphenamine and a bismuth preparation. Persistently positive Wassermann reactions were recorded in 6.6 per cent of patients with primary and secondary syphilis and in 22 per cent of those with latent, late and hereditary syphilis. Latency is a peculiar characteristic of the infection. It has been demonstrated at necropsy that a patient without clinical symptoms and with a negative serologic reaction may harbor intact spirochetes. Warthin [2] studied cases of such "cures" and found spirochetes and active lesions in the heart, aorta, testes, pancreas and liver. Biologic implantation tests have indicated that the spirochete, though dormant in the body of a carrier, may retain

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1. Stokes, J. H.; Cole, H. N.; Moore, J. E.; O'Leary, P. A.; Parran, T., Jr., and Wile, U. J.: Cutaneous and Mucosal Relapse in Early Syphilis and Its Differentiation from Reinfection, Ven. Dis. Inform. **12**:55, 1931.

2. Warthin, A. S.: The Persistence of Active Lesions and Spirochetes in the Tissues of Clinically Inactive or "Cured" Syphilis, Am. J. M. Sc. **152**:508, 1916.

its virulence and infectivity indefinitely. According to Warthin latent syphilis is more prevalent than is ordinarily suspected. He stated that the usual estimate, of from 10 to 15 per cent, is too low for the general incidence of syphilis and that 30 per cent expresses a more accurate indication of its prevalence. Warthin claimed that he never saw at postmortem examination a "cured" syphilitic patient. He described such patients as "damaged goods" and expressed the opinion that the damage is progressive. For proof of the eradication of the spirochete it is necessary to present a convincing series of careful necropsy studies on patients known to have had syphilis which was thoroughly treated. Thus far there has been no such proof. On the basis of clinical experience Fournier's dictum, "syphilis slumbers but does not become extinguished," still seems true.

Certain modern concepts of syphilitic infection may help to dispel some of the complacency which has settled around this question. It is now realized that syphilis is a chronic infection in which the invasion by spirochetes becomes generalized early (long before the chancre appears) and that the infection runs a prolonged and varied course, with a tendency to relapse. Individual variations occur according to the constitutional reaction of the patient to the invader. As Schindler [3] has aptly stated, success of treatment depends not on the serologic reaction, nor on a fixed scheme of treatment, but rather on the response of the constitution in the individual patient. There are constitutional differences in morphologic characteristics, function and even immunity which induce variations in response to the spirochete and to the treatment. A suggestive investigation in this direction was made by Nishiwara,[4] who expressed the opinion that there may be a relation between blood grouping and predisposition to disease. He found that the Wassermann reaction became negative with treatment in a higher percentage of patients whose blood belonged to group O or A than in those with blood of group B or A B. The physician who is aware of the importance of constitution as a factor in syphilis will treat the patient, not the spirochete or the serologic reaction. In patients with latent syphilis the serologic reaction may remain positive in spite of maximal treatment. In other patients the reaction becomes negative promptly but the infection may continue. Chatschaturjan [5] extirpated a lymph node from a syphilitic patient who had received six courses of treatment and

3. Schindler, K.: Die Konstitution als Faktor in der Pathologie und Therapie der Syphilis, Berlin, S. Karger, 1925.

4. Nishiwara, M.: The Relationship of Curability in Syphilis to Constitutional Serology, Lues: Bull. Soc. japon. de syph. **12**:2, 1935.

5. Chatschaturjan, G.: Latente Syphilis und Inokulation lymphatischer Drüsen, Dermat. Ztschr. **66**:315, 1933.

presented no symptoms and had negative serologic reactions. On scrotal inoculation of a rabbit with this material, a typical chancre developed. In the state of latency there is a balance between the invasive powers of the parasite and the immune reactions of the body. Bergel [6] concluded that agglutination, degeneration and lysis of the spirochete are induced by a lipolytic ferment from the lymphocytes, which constitute the main cellular defense against the spirochete. He explained the altered reactions in patients with tertiary syphilis by the altered nature of the spirochete after long exposure to the defenses of the body. The external lipoid layer of the spirochete has been digested, leaving the inner protein substance exposed. This acts as an antigen which induces the reactions typical of tertiary manifestations. Chesney [7] stated that acquired immunity to syphilis is a state of resistance which evolves comparatively slowly and is not always complete. It is based more on tissue reactions than on blood defenses. It is not the function of any one cell, nor is it attained by any known method of immunization. It evolves only as the result of reactive manifestations, though it may not depend on the persistence of infection for its maintenance. Finally, this symbiosis reaches the stage at which a balance has been attained between the host and the parasite, with mutual immunization, so that the organisms may remain viable for decades in the body of the host without increasing in numbers and yet are able to initiate an inflammatory reaction should conditions favor them. Theobald Smith [8] expressed the belief that the prolonged parasitism of the spirochete and the insidious, comparatively mild reactions induced only after a considerable period indicate that there is little that is foreign to the body in the parasite and that its metabolic activity is closely allied to that of the host. These are the conditions of a perfect symbiosis. This shows how difficult is the problem of chemotherapy, namely, to kill the invader without injuring the host. The syphilitic infection has been effectively treated clinically and experimentally by compounds of arsenic, bismuth, mercury, vanadium, platinum and gold. The term "specific treatment" is a misnomer. The present therapeutic armamentarium is a blunderbuss of nonspecific metal therapy, which occasionally wounds the host as well as the parasite. Arsphenamine does not destroy the spirochete in vitro, but some observers think that in the body an arsenoprotein is formed

6. Bergel, S.: Die Syphilis im Lichte neuer experimentell-biologischer und immuntherapeutischer Untersuchungen, Jena, Gustav Fischer, 1925.

7. Chesney, A. M.: Acquired Immunity in Syphilis, Am. J. Syph. **14**:289, 1930.

8. Smith, Theobald: An Attempt to Interpret Present Day Uses of Vaccines, J. A. M. A. **60**:1591 (May 24) 1913.

which is destructive to the parasite. In patients who are resistant to treatment an arsenic compound may form which is comparatively harmless to the parasite. The incidence of relapse in spite of vigorous therapy, latency and resistance to treatment may be due to one or more of the following factors suggested by Ingraham:[9] (1) A few of the parasites acquire tolerance for drugs; (2) the spirochetes are situated where a drug does not reach them; (3) the defensive powers of the host are defective; (4) the spirochete passes through a life cycle in which certain forms are resistant to drugs. A study of the comparative morphology and biology of the spirochete has revealed granular and budding forms. The presence of granular forms among the parasites of the spirochete group tends to indicate that this phenomenon is more than a mere degeneration. These facts have induced Marchand, McDonagh, Levaditi and others to postulate the existence of a complex life cycle for the spirochete. The existence of a granular or spore stage may be sufficient to explain resistance to treatment and a tendency to relapse. The actuality of such a life cycle has not been proved or confirmed.

One must therefore be content for the present to regard the spirochete as the sole cause of all the morbid changes associated with syphilis. When one takes into consideration the early generalization of the infection, the tendency to relapse, the uncertainties of modern "specific" therapy, the existence of latent and anergic infection and the little understood immunology of syphilis, a cautious, conservative and skeptical attitude as to the complete biologic cure of the infection seems justified. It is more accurate to speak of "arrest" than of "cure"; at least this attitude might discourage a too prevalent optimism and complacency. It is therefore illogical to set a time limit, such as from three to five years, after which a patient may marry presumably with assurance of safety. From an ideal eugenic standpoint the syphilitic person is undesirable marriage material. Should a syphilitic person marry and have progeny? If the syphilitic person insists on his biologic rights, tragedy is always possible, even with all the advantages of the highly developed modern treatment. A perusal of the best texts of modern syphilographers since 1910 indicates a tendency to prolong the duration of treatment and medical supervision of the syphilitic candidate for marriage. They recommend continuous treatment until the disease is clinically and serologically "cured," and then a lifetime of medical supervision. The course of syphilis is unpredictable even by the most skilled physician. Since the physician can at no time assure the patient that his body is free from spirochetes, is it safe at any

9. Ingraham, N. R., Jr.: The Life History of the Treponema Pallidum, Am. J. Syph. **16**:155, 1932.

time to assure the syphilitic man that he may marry without endangering his wife and offspring? At best there may be offered a prospect of a reasonable span of life with a minimum of complications and assurance of arresting the infection if it is properly treated in time. As concerns scientific medicine versus syphilis, the ancient struggle continues, with the hope that more profound studies in the biochemistry of the spirochete and more basic researches in immunology and chemotherapy may some day bring the realization of Ehrlich's therapia sterilisans magna for this stubborn infection.

# INCREASED REACTIVITY OF THE SKIN TO STAPHYLOCOCCUS TOXIN IN PATIENTS WITH LUPUS ERYTHEMATOSUS

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AND
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Since 1906 it has been known that filtrates from broth cultures of certain strains of staphylococcus contain a specific exotoxin.[1] On this fact, however, general attention was not focused until 1928,[2] when in Bundaberg, Australia, twelve children died after receiving injections of diphtheria antitoxin, which later was shown to have been contaminated with a toxin producing staphylococcus. In 1930 Burky[3] isolated similar staphylococci from various cutaneous lesions, and he, as well as others, has published studies dealing with this toxin.[4]

In order to determine the reactivity of the skin to this toxin shown by patients with various types of cutaneous diseases, we have performed intracutaneous tests on ninety-seven patients from the dermatologic dispensary. The test dose of toxin (Burky's) was 0.1 cc. of a 1:100 dilution, injected intracutaneously in the forearm. Reactions were observed forty-eight hours later.

The purpose of this paper is to report the unusual reactions of fifteen patients who had either active lupus erythematosus or scars resulting from former lesions of the disease.

Our results showed that patients with lupus erythematosus reacted much more frequently and violently than did the control group. Of the fifteen patients with lupus erythematosus tested, ten, or 66 per cent,

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From the Dermatological Clinic of the Johns Hopkins Hospital and the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

1. Kraus, R., and Pribram, E.: Ueber Staphylokokkentoxin und dessen Antitoxin, Wien. klin. Wchnschr. **19**:493, 1906.

2. Burnet, F. M.: The Exotoxin of Staphylococcus Pyogenes Aureus, J. Path. & Bact. **32**:717, 1929.

3. Burky, E. L.: Personal communication to the authors.

4. Burky, E. L.: Studies on Cultures and Broth Filtrates of Staphylococci, J. Immunol. **24**:93, 115 and 127, 1933; Studies on Cultures and Broth Filtrates of Staphylococci: Antitoxin Content of the Rabbit Serums Immune to Staphylococcus Toxin and Precipitin Reactions of Such Serums, ibid. **25**:419, 1933.

showed a reaction exceeding a total dimension[5] of 10 cm. Of the control group, consisting of eighty-two patients with various cutaneous diseases, such as verruca, eczema, scabies, dermatitis of various types, furunculosis, urticaria, acne vulgaris, chalazion, pityriasis rosea, ecthyma, impetigo and erythema multiforme, only 25 per cent showed a reaction exceeding 10 cm. It is interesting to note that the presence

*Data on the Results of Tests Made on Patients with Lupus Erythematosus* *

| Identification | Type of Lesion | Size of Reaction |
|---|---|---|
| Brown skinned Negro girl aged 17 | Chronic hypertrophic "butterfly" patch, present 1½ years | Local<br>15 + 5 cm. = 20 cm. |
| White man aged 28..... | Relapsing lesion in scar, resulting from old "butterfly" patch which had healed under gold therapy | Malaise<br>13 + 6.5 cm. = 19.5 cm.<br>12 + 7 cm. = 19 cm.<br>Tested twice |
| White woman aged 30..... | "Butterfly" patch of 4 years' duration | 9.5 + 7 cm. = 16.5 cm. |
| White woman aged 39..... | "Butterfly" patch of 5 years' duration | 6 + 4.5 cm. = 10.5 cm. |
| White man aged 27..... | "Butterfly" patch of 11 years' duration | 5 + 5 cm. = 10 cm. |
| White man aged 53..... | Recurrence in scar of "butterfly" patch; healed with therapy with a gold compound 4 years prior to testing | Fever, headache and chill<br>11 + 8 cm. = 19 cm. |
| White woman aged 33..... | History of "butterfly" patch healed by therapy with a gold compound several months prior to testing | 3 + 4 cm. = 7 cm.<br>(retested 4 weeks later, 7 + 4 = 11 cm.) |
| White woman aged 54..... | "Butterfly" patch and other subacute disseminate lesions; resistant to gold compounds | 8 + 5 cm. = 13 cm. |
| White woman aged 39..... | Acute disseminate lesions, with moderate constitutional reaction | 10 + 6 cm. = 16 cm. |
| White woman aged 39..... | Acute disseminate lesions; patient moribund at time of testing; diagnosis thought to be pellagra by some observers | 3.5 + 1.5 cm. = 5 cm. |
| White man aged 36..... | History of previous lesions; none at time of testing | 8.5 + 10.5 cm. = 19 cm. |
| White man aged 49..... | Several plaques about ears of 2 months' duration | 7 + 5 cm. = 12 cm. |
| White woman aged 47..... | Chronic plaques limited to scalp | 6 + 4 cm. = 10 cm. |
| White woman aged 32..... | Chronic plaques limited to scalp | 9 + 6 cm. = 15 cm. |

* Sixty-six per cent of the patients tested showed a reaction exceeding a total dimension of 10 cm. Only 25 per cent of eighty-two patients in a control group, with various diseases of the skin other than lupus erythematosus, showed a reaction exceeding 10 cm.

of an active lesion of lupus erythematosus was not necessary to provoke a strong reaction; patients with scars reacted just as strongly in some instances. The patient with the least reaction was moribund when tested.

This study indicates that patients with lupus erythematosus as a group react more intensely to staphylococcus toxin than do patients with other common cutaneous diseases. It is possible that the test may be of some aid in the diagnosis of questionable cases of the disease.

5. The dimension is obtained by adding the length and breadth of the reaction.

# Minor Notes

## TREATMENT OF IODINE BURNS WITH SODIUM THIOSULFATE SOLUTION

RICHARD L. SUTTON JR., M.D., KANSAS CITY, MO.

Tincture of iodine is a commonly used and valuable topical antiseptic, but when applied to some skins it can cause an acute dermatitis, which rapidly becomes vesicular and sometimes even ulcerates. It is well to know that a chemical antidote is readily available and strikingly effective.

Sodium thiosulfate in a weak aqueous solution is harmless and soothing as a wet dressing. It reacts with iodine in this fashion:

$$Na_2S_2O_3+4I_2+5H_2O \rightarrow 2\,NaI+2H_2SO_4+6HI$$

A patient, Mrs. W., aged 42, underwent an operative procedure wherein radium was inserted into the uterine cavity to inhibit abnormal bleeding. The usual surgical preparation included painting the external genitalia, the thighs and the buttocks with tincture of iodine. As soon as the patient recovered from the effects of the anesthetic, she complained of burning of these areas and informed her surgeon that on previous occasions severe reactions had followed the application of iodine to her skin. Although the excess amount of iodine had been removed with alcohol at the time of the operation and further effort was made at once to remove what remained, the irritation progressed and vesiculation occurred within twenty-four hours in spite of the application of zinc oxide ointment. At that time the severe dermatitis, which was limited sharply to the area where the iodine was applied, consisted of large and multilocular vesicles, containing clear yellow fluid, in confluent patches. A solution of sodium thiosulfate, 1:200 (one drachm [3.6 Gm.] to a quart of water [946 cc.]) was applied as a wet pack. This was comforting to the patient and was spectacular in relieving the inflammation. No new vesicles appeared, and in two more days the skin looked healthy and dry and was peeling as after a violent sunburn. The patient was certain that this was the quickest healing in her experience with iodine burns, for she had had them before.

A solution of sodium thiosulfate (1:200) applied on a cotton pad to my own skin on which tincture of iodine had been swabbed and allowed to dry removed all visible trace of discoloration from the iodine after six minutes. If to a few drops of tincture of iodine is added a sufficient amount of solution of sodium thiosulfate, the mixture instantly becomes clear, colorless and transparent. The blue stain on cloth produced by a combination of starch and iodine is similarly decolorized. A solution of sodium thiosulfate (1:200) is safe for wet dressings. I have used it frequently in the treatment of tinea and have never seen any harmful effects.

I thought for a time that this therapeutic idea was original with me, but Dr. L. A. Calkins tells me that he has known it for some time and that a student of his had the same inspiration several years ago. Webster[1] in his "Toxicology" suggests gastric lavage with a 5 per cent solution of sodium thiosulfate as an alternative to the use of starchy fluids in the treatment of poisoning by iodine taken by mouth.

Wet packs of sodium thiosulfate constitute specific treatment for iodine burns.

1308 Bryant Building.

1. Webster, R. W.: Toxicology, Philadelphia, W. B. Saunders Company, 1930, p. 387.

# Obituary

## JOSEF JADASSOHN
### 1863–1936

On March 24, 1936, dermatology sustained an irreparable loss through the death of Geheimrat Josef Jadassohn, emeritus professor of dermatology and syphilology of the University of Breslau. He died suddenly in Zurich, Switzerland, after an operation for acute abdominal obstruction.

Jadassohn was born in the small town of Liegnitz, Germany. He pursued his medical studies in the universities of Göttingen, Heidelberg and Breslau, receiving his degree in 1888.

From 1887 to 1892, he was the assistant to Albert Neisser in the dermatologic clinic of the University of Breslau. During these years a firm and lasting friendship was established between Neisser and Jadassohn, and they collaborated on many significant contributions in the fields of syphilis, gonorrhea, tuberculosis of the skin and atrophy of the skin and in the histopathologic investigations of cutaneous diseases.

In 1892, Jadassohn was appointed chief of the dermatologic division of the Allerheiligen Hospital in Breslau. Here he remained until 1896, and during these four years he contributed numerous valuable studies. Jadassohn's early contributions clearly showed a definite trend toward certain special problems of basic biologic significance, which later became the pattern of his life's medical work. His astounding knowledge of almost every aspect of medicine and biology, combined with his extraordinary breadth of vision, enabled him to exert great influence in bringing dermatology back into the domain of medicine. Jadassohn's subjects of investigation included such diverse interests as: the genesis of eczema; the morphology and immunology of tuberculosis of the skin, of tuberculids and of the dermatomycoses; the histology and genesis of congenital anomalies; gonorrhea; chancroid, and all forms of research in syphilis, as well as sociologic studies in the prevention of venereal disease, notably the legal regulation of prostitution.

The quality of this early work gained merited recognition, and in 1896, when only 33 years old, Jadassohn was called to the chair of dermatology at the University of Bern, Switzerland. He occupied this position until 1917, and it was during these years that he attained the stature of an international authority on almost every aspect of dermatology and venereology.

During these undisturbed and happy years in Bern, Jadassohn's accomplishments were prodigious. Today, many seemingly isolated and daring outposts of ultramodern dermatology are merely consolidating the advance made by the pioneer work of Jadassohn and his school. While in Bern, the sphere of Jadassohn's influence widened, not only because of his own work and that of his assistants and pupils but also because of his inspiration and guidance of illustrious disciples, among them Bruno Bloch and Lewandowsky.

In 1917 Jadassohn was called to Breslau to succeed Neisser. And in Breslau, with its richer material, superior equipment and large personnel, Jadassohn was able to increase the scope of his work and vastly to widen the sphere of his influence. His vitality and interests never slackened, and his achievements continued to surpass themselves to the last days of his life, long after he had been retired from the chair in Breslau (1931) after two special prolongations beyond the usual age limit. In Breslau, as in Bern, Jadassohn's teaching and example led to the development of distinguished disciples, such as M. Jessner, Martenstein, Biberstein, W. Frei, R. L. Mayer, St. Epstein, Lenhoff, Kogoj, Jordan, Truffi, Bizozzero, Dohi, H. N. Cole, Zwick, W. Freudenthal and Werner Jadassohn.

The published work of Josef Jadassohn is so voluminous and the subjects cover such wide and varied fields that the student can scarcely "read up" on any subject in dermatology and venereology without encountering some fundamental contribution from Jadassohn's pen.

It is obviously impossible to enumerate the achievements of this man of unique accomplishments and untiring zeal, nor is it possible to select, other than arbitrarily, certain contributions as being more worthy of mention than many others of equally preeminent value. It can never be forgotten that Jadassohn was the first dermatologist to apply objective experimental methods to the study of the immunology of the skin; the first to study cutaneous reactions and to apply a patch test; the first to study the precise influences of idiosyncrasy and allergy in the production of eczema, of drug eruptions, of tuberculids and of trichophytids, and the first fully to recognize the influence of immunobiologic effects on the morphology and course of many other dermatoses.

He elaborated many refinements in the modern diagnosis of gonorrhea and urged the general use of complement-fixation tests and of culture methods in the more accurate diagnosis of this disease.

And it is most likely that the employment of arsphenamine in the treatment of syphilis would not have survived the early accidents and the onslaughts of its opponents had not Neisser and Jadassohn stoutly and passionately championed the continued use of Ehrlich's drug in the face of the most bitter and often acrimonious opposition.

Jadassohn was considered by many to be the greatest authority of his time on such varied and diverse conditions as tuberculosis of the skin, the pyodermas, pellagra, the nevi, the dermatomycoses, drug eruptions and eczema, and his many works on these subjects have brought forth both clarity and new approaches.

Although he had never been in the tropics, he was a world-wide acknowledged authority on leprosy, and it is noteworthy that he was recently asked by the Brazilian government to inaugurate and to direct, as well as to organize, the research at the new Institute for the Study of Leprosy.

Jadassohn was the almost poetical creator of the majority of the dynamic problems of modern dermatologic research. The immunobiologic concepts of Jadassohn and Lewandowsky's law, of positive anergy and of localized sensitization and immunization are all his, and so also are the present classifications of the pyodermas, of the drug eruptions and of the tuberculodermas.

His incredible faculties of clinical and histologic observation and description are evidenced by the many sharply portrayed disease entities which bear his name, such as Jadassohn's blue nevus, Jadassohn's anetoderma, Jadassohn's granulosis rubra nasi, Jadassohn's psammoma, Jadassohn's dermatitis psoriasiformis et lichenoides and Jadassohn's lichen trichophyticus (lichenoid trichophytid).

In addition to possessing all these qualities of the scientist, Jadassohn was preeminently a physician, a truly remarkable diagnostician and therapeutist.

Jadassohn was not only the most eminent dermatologist but the recognized foremost venereologist of his time, and as such he was called on by the League of Nations to serve as the judge of serologic methods at the first international competition of serologists in Montevideo. He was a member of the Hygienic Commission of the League of Nations and chairman of the committees on the treatment of syphilis and on venereal diseases; he was also the president of the German Society for Combating Venereal Disease. Moreover, the German government officially called on Jadassohn to formulate the law for the control of venereal diseases which came into effect in 1927.

Besides holding innumerable other positions of responsibility and honor, Jadassohn was a director and perpetual general secretary of the German Dermatologic Society. He was editor of the *Archiv für Dermatologie und Syphilis* and of the *Zentralblatt für Haut- und Geschlechtskrankheiten* and co-editor of the *Dermatologische Wochenschrift* and of the *Klinische Wochenschrift*. He was honorary and corresponding member and fellow of innumerable dermatologic and other medical societies, including the American Dermatologic Association and the New York Academy of Medicine.

No mention of Jadassohn's achievements can omit the "Handbuch der Haut- und Geschlechtskrankheiten." This encyclopedic work—forty-one volumes—will stand as a monument to Jadassohn's labors as its guiding spirit and editor in chief. He also contributed several invaluable chapters.

Jadassohn the man, the personality, was as distinguished as Jadassohn the scientist. His integrity, his kindness, his indescribable charm, his delightful sense of humor and his genuine modesty—his character, in short—was a rare addition to his intellectual gifts.

He himself said that in his younger days he was given to outbursts of temper and was inclined to be intolerant toward those who did not, in his opinion, apply the proper standards of industry and scrupulousness to the work at hand. No traces of this choleric disposition remained noticeable during his later years. It was unique and profoundly touching to observe the adoration which Jadassohn's assistants felt for the "chief."

To epitomize the essential quality of this man's greatness, one can do no better than quote his son, who said: "My father is the personification of the sense of duty." And, in truth, duty was the motif of Josef Jadassohn's life: duty to his fellow man, duty to his craft and duty to his family. Humanity has lost a noble man, and dermatology, its greatest leader.

M. B. S.

## AN APPRECIATION

On a hot day in August 1911, while approaching the dermatology building of the old Inselspital at Bern, Switzerland, I ran into a somewhat short, stout man with mussed black hair and a very pleasing face. I asked him, in a halting German, where might I get in touch with the Herrn Professor Doktor J. Jadassohn. He immediately took me by the hand, in a kindly, fatherly way, and asked me who I was and for what I was desirous of seeing him, as he was Professor Jadassohn. That was my introduction to one of the finest, most lovable and kindliest men that it has ever been my experience to meet.

No one was idle in his clinic. Within a day or so he had submitted several problems to me, asking me to take my pick. I was given a stand in a well lighted passageway, for the old Inselspital did not boast of any elaborate group of buildings. They believed rather in specializing in brains, and the university could boast of the following galaxy—Sahli, in internal medicine; Kocher, in surgery; Langhans and Wegelin, in pathology; Kronecker, in physiology; Jadassohn, in dermatology; Kolle, in bacteriology, and others almost equally famous. I was furnished simply with a stool and a plain board stand for my microscope and sections, and I was told that I might go to work. Twice a day the staff made rounds in the hospital with the "chief," discussions being

held over patients and over conditions in which he was interested. While Jadassohn believed firmly in properly controlled experimental medicine, with human beings as well as with animals, he was one of the kindliest of persons. Many times I have heard him recommend to the chief of staff that certain persons be given an extra portion of bread and milk. There were several physicians outside his regular staff studying at the clinic. Included among them were two men from Japan, one from Russia, a woman physician from Stockholm, several Germans and another American besides myself. Time was never wasted in Jadassohn's clinic. The example furnished to all the assistants and to the outsiders by the "chief" was in itself sufficient. His delight was in a problem. He worked day in and day out. It was common knowledge in the clinic that when he took a vacation, which was always a short one, somewhere in the Swiss mountains or on a Swiss lake, he would take along a batch of papers, two or three boxes of slides and his microscope. He seemed to be interested in nothing except his beloved dermatology and the various problems connected with it.

At that period he had become especially interested in the various phases of immunology in relation to dermatophytosis and to tuberculosis—studies that, as time went on, were to make him world famous. It seemed to be his whole life.

One day his wife explained to me that they had some tickets for the Stadt Orchestra and that since Professor Jadassohn was not especially interested in music, though his brother had been the great pianist and composer Jadassohn, she would be happy if I would accept one of the tickets, and thereafter it was my especial pleasure throughout the winter to have one of his tickets for the Stadt Orchestra. The professor preferred to stay at home and work on some of his numerous problems.

While I have stated that he was always kindly, always interested in the various studies, he could be critical of results, and he was never satisfied until they actually crystallized into something very tangible. He had no time for a slacker. He would accept no short cut, and he did not believe in rushing into print. When my first paper was completed and I made the comment that I regretted very much handing in an untyped paper, I was told that if I cared to come to his home I might use his typewriter for that purpose; such little courtesies as this were shown to all in the clinic. No man who has ever worked with Jadassohn can have any but the kindliest recollections of this great man and scientist.

He never spoke ill of any one. Tension was rather high when I was in the clinic at the time of the Agadir episode, and although Jadassohn was a German I never heard him speak in any but the highest

words of admiration of the French school of dermatology. He was particularly an admirer of Brocq, of Darier and of Sabouraud.

Jadassohn furnishes an example for all the dermatologic world of a man interested in truth and in a medical science that has no bounds of race, color or creed. He was one of the great men of his time. As a teacher and investigator, he was unsurpassed, and men proud to acknowledge him as teacher are spread over the entire world.

H. N. C.

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A number of American colleagues, who were charmed by Jadassohn's personality on the occasion of his visit to the United States, expressed the opinion that the guest of the American Dermatological Association was displaying, if I may so put it, his "company manners." This opinion came to me as a surprise, because Jadassohn's conduct in America differed in no way from his unaffected, engaging simplicity of bearing as I observed it during a period of nearly ten years in the Dermatologische Universitäts-Klinik in Bern, Switzerland, where I received my fundamental instructions from the *praeceptor mundi in rebus dermatologicis*. At no time did Jadassohn "live on high in frigid dignity;" on the contrary, he taught by his daily example the principle of conduct which he professed and which one finds embodied in Goethe's imperative:

"Edel sei der Mensch,
Hilfreich und gut!"

It was in this spirit that Jadassohn gave to the assistants and to the research men in his clinic so freely of his time, from the day of their arrival, when he advised them how to find living quarters, until the time of their departure, when he provided them with letters of introduction to other clinics and celebrities.

In assigning research problems, he sought to discover the particular interests and specific qualifications of the student; he pointed out the terra incognita which the investigation was intended to explore; he assisted the search in the literature with references and reprints from his extensive private library. In making the rounds of wards and in discussing cases, Jadassohn developed the essential characteristics with the ease and clearness which are the enviable attributes of a great teacher. The skill and simplicity with which he interpreted the signs and symptoms of a disease in terms of a dynamic pathology are unforgettable. He instilled in his students a deep interest in unusual and rare conditions, because, as he declared, they so frequently lead to discoveries. What he taught us will remain in our memory.

As a man, Jadassohn was quiet and democratic, with a ready sense of lively humor. But though he was *suavis in modo*, as a critic he was *fortis in re*. His modesty was so great that his publication on Ehrlich's arsenical was prefaced by the aphorism of the Koenigsberg philosopher: "Wenn die Könige bauen, haben die Kärrner Arbeit" ("When the kings build, the truckmen have work"). Jadassohn's kindness inspired affection, and his high ideals fostered universal respect in all who came in contact with him.

To all those who have enjoyed the privilege of working with Jadassohn at any period of his life, but perhaps most of all to those who were with him in Bern, as I see it in retrospect the happiest period of Jadassohn's life, his death comes as a reminder of a time when his stimulating interest and enthusiastic desire to help were a part of their own best and busiest days!

The infrangible, enduring bond between Jadassohn and those who worked with him is one of the most remarkable tributes to his character. In the singularly appropriate words of Ruskin: "That man is richest who, having perfected the functions of his own life to the utmost, has also the widest helpful influence over the lives of others!"

K. G. Z.

# Correspondence

## SODIUM THIOSULFATE IN THE TREATMENT OF ARSENIC POISONING

*To the Editor:*—In a recent article entitled "Study of a Group of Handlers of Arsenic Tri-Oxide," by H. G. Irvine and D. D. Turnacliff (ARCH. DERMAT. & SYPH. **33**:306 [Feb.] 1936), mention was made that no book on toxicology or industrial medicine, published even as late as 1934, includes mention of the use of sodium thiosulfate for the treatment of industrial arsenic poisoning. This point is well taken; however, in the column of Queries and Minor Notes in *The Journal of the American Medical Association* (**99**:1374 [Oct. 15] 1932), the use of this drug was advised in a series of cases of dermatoses in laborers who sprayed potato plants with a combination of calcium arsenite and paris green. This reference was used in my recent paper entitled "Industrial Dermatoses: Treatment and Legal Aspects: Review of Recent Literature," published in the *Journal of Industrial Hygiene* (**17**:138 [July] 1935).

JOHN GODWIN DOWNING, M.D., Boston.

# Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

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SPINDLE-CELL EPIDERMOID CARCINOMA. HAYES E. MARTIN and FRED W. STEWART, Am. J. Cancer **24**:273 (June) 1935.

The authors describe a malignant metastasizing epidermoid carcinoma of the skin and mucosa. Eight cases are reported, most of which occurred in, or adjacent to, the lip. The lesions resembled carcinoma clinically and exhibited sarcomatous features histologically. Etiologic consideration is given particularly to roentgen and also to radium irradiation, cautery, wounds and other traumas resulting in inflammatory cutaneous changes in fibrous tissue. Cautery or endothermy applied to tissue exhibiting scars due to radiation is credited for the development of spindle cell carcinomas. These lesions are not radiosensitive and require wide and deep excision.

THE MALIGNANT TUMORS OF THE PERIPHERAL NERVES. ARTHUR P. STOUT, Am. J. Cancer **25**:1 (Sept.) 1935.

The most common primary malignant tumors of the peripheral nerves are fibrosarcomas; the majority of these occur in patients with Recklinghausen's disease and apparently originate in preexisting neurofibromatous nodules. Surgical removal is frequently followed by recurrences, and in 20 per cent of the cases metastasis occurs. These lesions are very radioresistant. Less frequent, and likewise of mesoblastic origin, are neurofibromas. Malignant tumors of peripheral nerves of epithelial origin are rare. Stout disputes Ewing's contention that the majority of spindle cell sarcomas of the skin are of neurogenic origin.

ON THE DYSONTOGENETIC ORIGIN OF BASAL-CELL CARCINOMA. JOSEPH MCFARLAND, EMMETT CICCONE and JOSEPH GELEHRTER, Am. J. Cancer **25**:273 (Oct.) 1935.

A review of the recorded localizations of basal cell carcinomas in two separate groups of hospitalized patients and a comparison of these observations with those of Glasunow indicated an anatomic distribution similar to that of sequestration dermoids and mixed tumors and a conformity with the localization of embryonic facial fissures. The authors therefore conclude that basal cell carcinomas may be dysontogenetic tumors which originate in imperfections in the closure of the embryonal facial fissures.

THE DIFFERENTIAL MORTALITY FROM CANCER IN THE WHITE AND COLORED POPULATION. S. J. HOLMES, Am. J. Cancer **25**:358 (Oct.) 1935.

Holmes states that racial differences apparently account for marked variations between the death rate from cancer in white patients and that in Negro patients. The younger age groups showed higher mortality among Negroes than among white patients, and the higher age groups showed the opposite. Among patients with cancer of the skin and buccal tissues the death rate was much higher for the white race, particularly in the age group beyond 50.

TUMORS OF THE PERIPHERAL NERVES. CHARLES F. GESCHICKTER, Am. J. Cancer **25**:377 (Oct.) 1935.

This paper includes a study of forty cases of Recklinghausen's disease. The tumors observed in cases of Recklinghausen's disease are usually undifferentiated

benign lesions of the nerve sheath that may recur after removal and that may undergo malignant change. They usually consist of reticulated myxomatous tissue and have been referred to as fibromyxomas, fibroneuromas and myxoid neurinomas. The malignant lesions are usually sarcomas of the nerve sheath. Recklinghausen's disease is a congenital disturbance of the nerve sheaths resulting in the formation of multiple subepidermal nodules along the distribution of the peripheral nerves, tumors of deeper nerve trunks and roots and disturbances in pigmentation. It may be complicated by meningeal tumors, angiomas, lipomas, plexiform neuromas, spina bifida and localized hypertrophy of a limb. Fifteen of the forty cases studied terminated fatally because of the occurrence of sarcomatous and other tumors.

THE RELATIONSHIP BETWEEN VASCULARITY AND THE REACTION TO RADIUM OF SQUAMOUS EPITHELIUM. M. G. SEELIG, C. T. ECKERT and Z. K. COOPER, Am. J. Cancer **25**:585 (Nov.) 1935.

On the basis of experiments on rabbit ears the investigators concluded that changes in the vascularity of the skin do not alter its radiosensitivity and that the latter is an inherent quality of the cell, variable in different subjects.

A CASE OF SCHÜLLER-CHRISTIAN'S DISEASE UNDER OBSERVATION FOR NINE YEARS. W. A. HANSON, L. H. FOWLER and E. T. BELL, Am. J. Cancer **25**:768 (Dec.) 1935.

The authors state that Schüller-Christian's disease is a form of xanthomatosis in which the xanthomas develop in various organs, especially in the bones of the skull. In the case reported there was involvement of the flat bones of the skull, the lumbar portion of the spine, the ilia and lymph nodes. Cutaneous lesions were not reported.

FOERSTER, Milwaukee.

CONGENITAL SYPHILIS IN CHILDREN. F. R. SMITH, Am. J. Syph. & Neurol. **19**:532 (Oct.) 1935.

The material for this paper was obtained from the records of 991 children with congenital syphilis seen in the Harriet Lane Home for Invalid Children, the pediatric department of the Johns Hopkins Hospital, from the opening, in 1914, to June 30, 1934.

The results of treatment in 279 patients with early congenital syphilis were analyzed. These patients were under surveillance for a minimal period of two, and a mean period of six and two-tenths, years. Congenital syphilis is a material factor in the cause of death in children under 6 months of age, the number of deaths due to the disease decreasing rapidly after that age. The beneficial results of prenatal treatment on the child are in direct proportion to the amount of treatment given the mother. "Ultimate satisfactory clinical response" to treatment is in direct proportion to the age at which treatment is started and to the amount of treatment. The incidence of Wassermann-fastness increases as the start of treatment is delayed. Its presence increases the danger of relapse and progression. Inadequate treatment does not delay the time of the appearance of lesions indicating a relapse, but adequate treatment prevents them.

All forms of early congenital syphilis are readily amenable to modern methods of treatment with the exception of clinical neurosyphilis.

THE DIAGNOSIS OF INFANTILE CONGENITAL SYPHILIS DURING THE PERIOD OF DOUBT. N. R. INGRAHAM JR., Am. J. Syph. & Neurol. **19**:547 (Oct.) 1935.

Infants born of mothers with latent or partially treated syphilis are apparently healthy at the time of birth in such a large percentage of cases that it is difficult or impossible to recognize early congenital syphilis, when it is present, by ordinary routine examination. About one half of such children are infected.

It is essential to give the new-born infant of a syphilitic mother the benefit of special study.

As regards children of mothers with latent or partially treated syphilis, physical examination of the child at the time of birth is of diagnostic value in not more than from 2 to 3 per cent of the cases; serologic tests of the blood are of some diagnostic worth in about 15 per cent, but of absolute diagnostic value in only about 1 or 2 per cent, of the cases. Dark-field examination of the umbilical vein, will, in routine practice, reveal spirochetes in about 20 per cent of infants born of syphilitic mothers while roentgen examination of the long bones will show an osteochondritis in about 30 per cent of such infants at the time of birth.

A combination of all these methods of study will detect in the first week of life at least half, and by the end of the second month about three fourths, of the babies ultimately proved to be syphilitic. There is, then, in the period of doubt, no absolutely reliable method of diagnosis, but a careful study of each child and the application of such diagnostic methods as do exist make it possible to detect infantile congenital syphilis while it is yet "curable" and keep the mortality and morbidity rate among the syphilitic infants at low figures.

It seems rational that an infant suspected of having contracted syphilis in utero from his mother should not be treated for this disease until he has been proved to have it and that he should not be subjected to antisyphilitic treatment merely because there is danger of his having the disease. REUTER, Milwaukee.

SYPHILIS: A REVIEW OF THE RECENT LITERATURE. JOSEPH E. MOORE, Arch. Int. Med. **56**:1015 (Nov.) 1935.

Moore gives abstracts of articles published in 1934 and up to July 1935; some of these have been abstracted and others have been published in earlier issues of ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY. JAMIESON, Detroit.

SYPHILITIC AORTITIS IN CHILDHOOD AND YOUTH: REPORT OF TWO CASES WITH SUDDEN DEATH. ROBERT F. NORRIS, Bull. Johns Hopkins Hosp. **57**:206 (Oct.) 1935.

Norris reports 2 cases of syphilitic aortitis. One occurred in a 9 year old Negro girl and the other in a white boy aged 17. There was considerable evidence that the lesions were of congenital origin. The patients were the only ones among 14,000 patients examined at autopsy at the Johns Hopkins Hospital who showed pathologic changes suggestive of congenital syphilis of the aorta. In each case death was comparatively sudden. NELSON PAUL ANDERSON, Los Angeles.

THE ADRENAL PROBLEM. FRANK A. HARTMAN, Endocrinology **19**:633 (Nov.-Dec.) 1935.

In a review of recent work on the adrenal glands Hartman points out that in experimental chronic adrenal insufficiency produced by the administration of inadequate doses of adrenal cortex extract in adrenalectomized animals pigmentation has been observed only in monkeys. The absence of pigmentation in other animals is of interest because hyperpigmentation is such a striking feature of Addison's disease. LYNCH, St. Paul.

THE INTRADERMAL REACTION FOR CHANCROIDS WITH CHANCROIDAL BUBO PUS. H. N. COLE and E. A. LEVIN, J. A. M. A. **105**:2040 (Dec. 21) 1935.

Cole and Levin confirm the observations of previous workers as to the specificity of an antigen prepared from the pus of chancroidal buboes and used as an intradermal diagnostic test for chancroid. The antigen is prepared from a chancroidal bubo which has been proved to be chancroidal by the presence of Ducrey's bacillus either in the primary lesion or in pus from the bubo. The patient from whom the material is obtained for the test is required to have a negative Frei reaction and the antigen prepared must not produce reactions in

patients with lymphogranuloma inguinale. Negative reactions to the intracutaneous test were noted in patients with lymphogranuloma inguinale, gonorrhea, granuloma inguinale and pulmonary tuberculosis, in patients with early and with late stages of syphilis, in pregnant women, in patients with various cutaneous diseases, including mycotic infections, and in patients with varied conditions who were in wards of the medical service. Of fifty-two patients who had positive reactions to the intradermal test, five had no evidence of active infection or history of such an infection; twenty-two harbored the organism; five were known to have had a previous infection, and twenty had a history of previous venereal ulceration and suppurating buboes. There was an average latent period of five weeks from the presence of the ulceration to the production of a positive reaction. The presence of a chancroidal bubo seemed to increase the rapidity and intensity of the reaction to the intracutaneous test. Positive and negative reactions noted from the injection of the pus antigen used in the investigation closely paralleled those obtained with a stabilized streptococcus vaccine; the latter gave a more clearcut and stronger reaction. The authors conclude that the allergic response to the intracutaneous test in a patient with a chancroidal infection persists for at least from thirty to thirty-four years and probably for life. The therapeutic results obtained by subcutaneous injection of the material were good in several instances of resistant infection.

CAUSAL THERAPY OF CONTACT DERMATITIS. THEODORE CORNBLEET, J. A. M. A. **105**:2062 (Dec. 21) 1935.

Cornbleet mentions that in establishing the diagnosis of contact dermatitis it is important to note the distribution and morphology of the lesions in order to detect the agent responsible for the dermatitis. It is necessary to obtain thorough information as to the patient's work, activities and environment. It is also helpful to have some knowledge of the common irritants and allergins to which the patient might be exposed, as well as of the agents capable of producing the greatest number of sensitivities. All materials which are suspected should be applied as patch tests, suitable concentrations being used, to the normal skin of the patient. The interpretation of the results of the test requires experience, and attention is drawn to false negative and false positive reactions. The final conclusion as to the cause of the dermatitis is the demonstration of clinical cure when a suspected allergin is removed from the patient's environment. Desensitization is occasionally successful, and in some cases a cure may be effected by merely preventing contact between the offending allergin and the patient.

RADIOTHERAPY (ROENTGEN RAYS; RADIUM). ARTHUR U. DESJARDINS, J. A. M. A. **105**:2064 (Dec. 21); 2153 (Dec. 28) 1935.

Among inflammatory conditions notably amenable to roentgen therapy may be mentioned furuncle, carbuncle, acute simple adenitis, acute parotitis, abscess and cellulitis of soft tissues, onychia and paronychia, mastitis, sinusitis, mastoiditis and delayed resolution of lobar pneumonia.

The main factor in the action of the rays in these acute inflammatory conditions consists in the destruction of the infiltrating leukocytes. It seems likely that the destruction of the leukocytes liberates the antibodies and other protecting substances previously elaborated within the cells and thus makes these substances more readily available for defensive purposes than when they were in the intact cells. Chronic inflammations such as tuberculous adenitis and peritonitis, actinomycosis and blastomycosis are also amenable to roentgen therapy. The mode of action in the latter disease depends on the degree of leukocytic infiltration and also on the proportion of connective tissue and of degenerative material and calcium.

Desjardins classifies tumors according to the degree of their radiosensitivity. The first class embraces radiosensitive tumors; the second class, moderately radio-

sensitive tumors, and the third class includes radioresistant tumors. Basal cell epithelioma is considered to belong in the first class, whereas prickle cell epithelioma is placed in the second class. Melanomas are the most resistant of neoplasms.

Each variety of cell in the body is specifically sensitive to roentgen rays or to radium. According to Desjardins, cells may be classified in the order of their degree of sensitiveness as follows: (1) lymphoid cells; (2) polymorphonuclear leukocytes and eosinophils in the blood or tissues; (3) epithelial cells; (4) endothelial cells; (5) connective tissue cells; (6) muscle cells; (7) bone cells; (8) fat cells; (9) nerve cells. The skin may tolerate with impunity a considerable amount of roentgen rays, but when the limited tolerance is exceeded a series of reactive changes occur. Roentgen ray dermatitis is manifested by early and late effects. The mucous membranes are of approximately the same sensitivity as the skin. Desjardins does not believe that there is any stimulating effect from roentgen or radium radiation. Stimulating effects apparently resulting from exposure to small doses of roentgen rays indicate exceptional radiosensitiveness. In the treatment of inflammatory disease by means of roentgen and radium radiation, small or moderate doses only are employed. The more acute the lesion, the smaller is the dose of rays required. If the treatment is given early during the stage of leukocytic infiltration and before suppuration has set in, the results are apt to be better than if given later. Pain is frequently relieved by radiation.

Artificial Fever Therapy of Syphilis. Walter M. Simpson, J. A. M. A. **105**:2132 (Dec. 28) 1935.

Simpson treated 175 patients with syphilis by means of artificial fever therapy. The apparatus used is known as the Kettering hypertherm and consists of an insulated cabinet in which the nude patient lies on an air mattress. The interior is air conditioned, the temperature being regulated by a thermostat. The humidity is also kept constant, which adds to the safety and comfort of the patient. High frequency currents are not used. Simpson agrees with other observers as to the value of artificial fever therapy in the management of neurosyphilis. His results compare favorably with those obtained by the use of malarial therapy. The advantages of artificial fever therapy over malarial therapy are that the former does not require hospitalization, is less time consuming and involves less hazard to the patient. Patients with primary or early secondary syphilis also received artificial fever therapy, and the improvement obtained leads Simpson to state that this method may be a useful adjunct to chemotherapy in the treatment of early syphilis. He expresses the belief that artificial fever therapy fortifies and intensifies the action of antisyphilitic chemotherapeutic agents.

Inguinal Gland Metastases in Carcinoma of the Penis. Benjamin S. Barringer, J. A. M. A. **106**:21 (Jan. 4) 1936.

Barringer reported on 100 cases of carcinoma of the penis which he abstracted and correlated from the records of the Memorial Hospital for the Treatment of Cancer and Allied Diseases in New York. In 63 cases only the primary lesion developed. In 37, or a little more than one third of the patients, metastases to the groin occurred. Nine of these 37 patients were still living at the time the report was made; the remaining 28 died of carcinoma and 1 of these lived more than three years after the initial observation. One half of those who died did so in the first year. From his study, Barringer concluded that inguinal metastases are exceedingly difficult to cure. He suggests that the primary lesion should be surgically removed as the first step in treatment. Removal of material for biopsy by aspiration of the inguinal nodes is imperative, and if cancer is present a combination of external irradiation and implantation of radon seeds through an incision and under vision seem to offer the best outlook.

Lewis, New York.

LUPUS CARCINOMA: REPORT OF AN UNUSUAL CASE OF CARCINOMA FOLLOWING INJURY AND IMPLANTED LUPUS VULGARIS ON THE LEFT UPPER EXTREMITY. HERMAN GOODMAN, J. Indust. Hyg. **17**:276 (Nov.) 1935.

Goodman describes a case of lupus vulgaris of the left elbow immediately following a minor occupational trauma; neoplastic changes revealed by microscopic examination took place within six months of the time of occurrence of the trauma. After excision and skin graft the lesions of lupus recurred. Goodman suggests that the tuberculous process developed as a result of the fact that the patient's wound was dressed with the soiled handkerchief of a fellow worker.

WIEDER, Milwaukee.

FUNGUS DISEASES OF THE SKIN. ARTHUR M. GREENWOOD, New England J. Med. **213**:363 (Aug. 22) 1935.

Laboratory examinations are the final resort in the diagnosis of diseases due to fungi. The procedure for examination of epidermal scales is simple but time consuming. According to Greenwood, the "mosaic" fungus is not a fungus. Biologic investigations along the lines of sensitization and allergic reactions are briefly discussed. Exacerbation of a chronic fungous infection of the skin by industrial conditions and fungous infections superimposed on industrial dermatitis have been held to justify requests for compensation as causes of industrial disability in Massachusetts. A fairly complete but brief discussion of therapy is presented; the highly irritating action of most proprietary remedies is emphasized. The location of the infection should be kept in mind when therapy is instituted. The two fundamental factors are emphasized: (1) the elimination of articles of clothing which encourage the growth of fungi, such as wool socks, heavy boots and gloves, wool underwear and athletic supporters; (2) the avoidance of irritating medication. The acute vesiculopustular reactions are best treated by application of wet dressings during the day; at night ointment containing salicylic acid and sulfur and dusting powder containing boric and tannic acid should be used. The subacute and chronic types are best treated by ointment containing salicylic acid and sulfur, Whitfield's ointment, carbolfuchsin paint or solutions of iodine. Roentgen irradiation produces favorable results, but it must be used with great care. Ethyl iodide should not be used by inexperienced persons nor be employed in unselected cases.

The following factors play a part in reinfection: (1) lack of immunity; (2) ease with which fungi can grow on all fabrics and material used as clothing; (3) ability of fungi to grow on débris and slime which accumulate on various floors; (4) increasing use of gymnasiums and habit of exercising in unsterilizable socks and body clothing; (5) lack of cooperation by authorities in charge of public baths and gymnasiums; (6) incomplete disinfection by standard laundry procedures.

Prophylaxis should include the daily scrubbing of floors with soap powder, the wearing of unlined rubber bathing shoes, the prohibition of walking barefoot, the use of sterilizable cotton garments, and, as the most important factor, the cleanliness of the feet and folds of the body.

APPEL, Boston.

IMPROVEMENT OCCURRING IN CASES OF CALCINOSIS UNIVERSALIS IN CHILDREN. F. PARKES WEBER, Brit. J. Dermat. **47**:400 (Oct.) 1935.

The author reports the occurrence of calcinosis universalis in three girls; diminution of the calcareous deposits was shown by roentgen examinations. The treatment included the use of iodides, ketogenic diet and disodium phosphate.

OCCUPATIONAL ARGYRIA. J. M. HARKER and DONALD HUNTER, Brit. J. Dermat. **47**:441 (Nov.) 1935.

The authors report sixteen cases of occupational argyria; in seven cases the condition was generalized, and in nine it was localized. All the patients in whom

the condition was generalized except one who handled fulminate of silver were employed in the manufacture of silver nitrate. The nine patients with localized argyria were silversmiths.

The generalized argyria occurred in workmen who handled some silver compound, and the eruption resulted from inhalation and ingestion of the silver salt. The localized form occurred in workmen who handled metallic silver, and the eruption was due to accidental tattooing. Generalized argyria is frequently associated with ophthalmologic changes. These consist of a silver staining of the conjunctiva, especially of the inner canthus. The pigment is situated chiefly in Descemet's membrane. To a lesser extent, the same type of ocular pigmentation may occur as the result of the prolonged use of drops containing organic compound of silver. Nine illustrations, five of which are colored, accompany the article.

POST-TRAUMATIC ANGIOMA. F. FÖLDVARI, Brit. J. Dermat. **47**:463 (Nov.) 1935.

The occurrence of angiomas in a child, 8 years old, following an injury to the nape of the neck is reported. Multiple grouped bluish-red angiomas from the size of a lentil to that of a pea appeared at the injured site in ringlike formation. The skin in the affected area was unchanged. There was no recurrence of the lesions after they were destroyed by electrocoagulation.

Histologic examination showed that the greatest changes occurred in the corium. These consisted in many newly formed capillaries lined with swollen endothelial cells. There was no evidence of inflammation or infiltration.

WIEN, Chicago.

THE PRESENCE OF LEISHMANIA DONOVANI IN THE NASAL SECRETION OF CASES OF INDIAN KALA-AZAR. H. E. SHORTT and C. S. SWAMINATH, Indian J. M. Research **23**:437 (Oct.) 1935.

Using ordinary smears of nasal secretion stained by Giemsa's stain, Shortt and Swaminath were able to demonstrate Leishmania Donovani in two of four cases of Indian kala-azar, thus confirming previous observations of Forkner and Zia.

They advise that in searches for the organism several slides be examined, as some may give negative, and only one may give positive, results.

LEPROSY: A REPORT OF TWENTY-SEVEN CASES TREATED WITH ANTHRAX VACCINE. J. N. ROUSSEL, J. Trop. Med. **38**:133 (June 1) 1935.

In treating patients with leprosy Roussel used a vaccine containing attenuated living anthrax bacilli, beginning with 0.125 cc. and doubling the dose until a dose of 2.5 cc. was reached. A total of 30 cc. was given each patient, the injections being given at intervals of five days.

No change was noted in the lesions until about two or three months after the injections were discontinued. In two thirds of the cases the lesions gradually faded away, the anesthesia being the last symptom to disappear. In the remaining cases a nodular, multiform erythema developed, the leprous lesions became painful and there was a marked elevation of temperature for three weeks. The nodules and then the leprous lesions disappeared. The anesthesia also slowly disappeared.

Of the twenty-seven patients treated with the anthrax vaccine, seventeen had maculo-anesthetic leprosy, nine tubercular leprosy and one a mixed type of the disease.

Eighty-eight per cent of the patients with maculo-anesthetic leprosy are apparently well; those with tubercular leprosy were not benefited; the one with the mixed type of the disease is also apparently well.

Emphasis is laid on the statement that the use of this vaccine is urged only for the treatment of maculo-anesthetic leprosy.

Brief reports of five cases are given. JAMIESON, Detroit.

THE ACTION OF CONCENTRATED SPLENIC EXTRACT IN CASES OF CERTAIN DERMATOSES (ECZEMA AND URTICARIA) AND OF ASTHMA. P. VALLERY RADOT and P. BLAMOUTIER, Paris méd. **2**:457 (Dec. 8) 1934.

Among forty-nine patients with eczema treated with splenic extract from which most of the albumin had been removed, twenty-one were cured, in twelve improvement was noted, in eight results were inconclusive and in eight no benefit resulted. Among thirty-five patients with urticaria there were eight complete cures, and in seven improvement or temporary benefit was noted. Among seventeen patients with asthma, four showed improvement, and in two of these there was almost entire relief from asthmatic symptoms.

The authors offer no explanation of their results but restate the theory of Schiff, that the spleen secretes an enzyme which changes trypsinogen into trypsin, and that of Paul, that eczema is caused by a defective metabolism of albumin due to insufficiency of the aforementioned enzyme. Introducing splenic extract into the circulation makes up for this deficiency.

LESLIE, Evanston, Ill. [AM. J. DIS. CHILD.]

OBSERVATIONS ON THE SARCOIDS (BESNIER-BOECK'S SYNDROME) AND ON SOME RETICULO-ENDOTHELIAL MANIFESTATIONS OF THE SKIN. A. CROSTI, Gior. ital. di dermat. e sif. **76**:975 (Aug.) 1935.

Crosti calls attention to the fact that in some cases of Besnier-Boeck's syndrome there are hyperplastic granulomatous lesions of pseudoleukemic character which warrant the interpretation given by Schaummann, that the condition is a benign lymphogranulomatosis, and by Pautrier, that it is a systematized reticulohistiocytosis. Three cases are reported to substantiate these views.

CONTRIBUTION TO THE STUDY OF CUTANEOUS SARCOIDS. G. RADAELI, Gior. ital. di dermat. e sif. **76**:1000 (Aug.) 1935.

Radaeli reports a case of lupus pernio of both ears, the disease affecting the auricular canal and the tympanic membrane on one side as well as both auricles. The patient also presented dermo-epidermic nodules on both hands, enlargement of the lymphatic glands and of the tonsils and a tumor of the spleen. The roentgen examination of the chest showed chronic fibrotic tuberculosis. Radaeli concludes that the tuberculous nature of this syndrome has not been sufficiently proved.

THE SARCOID TYPE OF LEPROSY. F. LISI and F. SEBASTIANI, Gior. ital. di dermat. e sif. **76**:1030 (Aug.) 1935.

A case of leprosy is reported in which the clinical as well as the histopathologic picture resembled closely that of cutaneous sarcoids. The diagnosis was based on the presence of leprosy bacilli in the lesions.

THE BENGAL ROSE TEST IN CERTAIN DERMATOSES, ESPECIALLY IN CASES OF ECZEMA. G. ROCCHINI, Gior. ital. di dermat. e sif. **76**:1076 (Aug.) 1935.

The study of the hepatic function by means of the bengal rose test was carried out in fifty-seven patients with cutaneous diseases. The conclusions reached by the authors are as follows: In the majority of the cases of occupational eczema of external origin, the hepatic function was normal; in a high percentage of cases of eczema of unknown, presumably of internal origin, the hepatic function was more or less seriously affected. In cases of psoriasis the test gave normal results.

THE ETIOPATHOGENESIS OF ERYTHEMA MULTIFORME IN PORADENITIS INGUINALIS. A. MIDANA, Gior. ital. di dermat. e sif. **76**:1091 (Aug.) 1935.

Midana reports a case of erythema multiforme in a patient suffering from poradenitis inguinalis. The virus was present in the cutaneous lesions. In view

of his observations Midana interprets the cutaneous lesions as the result of an allergic reaction due to localization of the virus in the cutaneous tissues by metastasis.

[This interpretation is at variance with the present conception of bacterial allergy in America. Allergic manifestations are regarded as reactions occurring at a distance from the focus of infection, and the offending agent is never recovered from them. Abstractor.]

THE FREI REACTION IN PORADENITIS INGUINALIS. LUIGI PERUCCIO, Gior. ital. di dermat. e sif. **76**:1098 (Aug.) 1935.

Peruccio has observed cases of undoubted poradenitis inguinalis in which the intradermal reaction to the Frei test was negative. He interprets these negative reactions as the result of an anergic condition of the patient or as due possibly to a recent infection in which the defensive mechanism has not reached full development. Further tests are indicated during the course of the disease. [The term "poradenitis inguinalis" has been discarded in America; the name "lymphogranulomatosis inguinalis" is still in use, but there is a tendency to accept the denomination "lymphopathia venerea" proposed by Wise and Sulzberger. Abstractor.]

COMPLEMENT-FIXATION TEST IN PORADENITIS INGUINALIS. L. PERUCCIO, Gior. ital. di dermat. e sif. **76**:1101 (Aug.) 1935.

Complement-fixation tests with antigens prepared from fresh pus in various dilutions gave uniformly negative results in fifteen cases.

CHEILITIS GLANDULARIS AND SIMILAR CONDITIONS. JOSE J. PUENTE, Rev. méd. latino-am. **20**:937 (June); 1061 (July) 1935.

In this monograph, which covers the subject thoroughly, Puente reports fifty-two cases of cheilitis glandularis. The condition is differentiated from cheilitis glandularis apostematosa of Volkmann, the latter being, according to Puente, a complication developing on the "simplex" type of cheilitis which he describes. Cheilitis glandularis simplex is characterized by enlargement of the orifices of heterotopic salivary glands situated on the lower lip. It is found in about 3 per cent of the patients attending the clinics for patients with cutaneous diseases in Buenos Aires, Argentina, but it occurs mostly among Spaniards residing in the northern part of the country. The condition may remain unknown for a long time, and the patients are, for the most part, entirely unaware of its presence. Several cases of malignant degeneration have been reported in which the epithelial growths seemed to have developed at the expense of the glandular tissue involved. Cheilitis glandularis simplex may be considered as a precancerous condition.

PARDO-CASTELLO, Havana, Cuba.

THE MORPHOLOGY OF THE INTERNAL RETICULUM (GOLGI-KOPSCH APPARATUS) OF THE CELLS OF HUMAN SEBACEOUS GLANDS. N. MELCZER, Dermat. Ztschr. **71**:129 (May) 1935.

The Golgi-Kopsch apparatus can be shown in the form of a network by means of special impregnation methods. In the matrix cells of the sebaceous glands this network is seen to be closely set around the nucleus. In the mature cells undergoing necrobiosis the network is scattered through the cytoplasm.

NEW FINDINGS CONCERNING FURUNCLES AND FURUNCULOSIS. M. M. LEWIN, Dermat. Ztschr. **71**:197 (June) 1935.

Lewin obtained cultures of staphylococci from furuncles of patients with a single furuncle and with furunculosis, from apparently normal skin of patients with furunculosis, from patients with weeping dermatoses, and from persons without cutaneous lesions. The virulence of these cultures was determined by intracutane-

ous injections into the skin of rabbits, the reactions being graded according to their severity and duration. Cultures obtained from the furuncles themselves were most virulent, in a number of instances causing necrotic and pustular reactions. Cultures from normal skin of patients with furunculosis and from areas of weeping dermatoses were somewhat less virulent, and those from patients without cutaneous conditions were only slightly virulent. The author concludes that a virulent strain of staphylococci rather than a reduced resistance is responsible for the development of furuncles.

HEAT URTICARIA. W. BARTELS, Dermat. Ztschr. **71**:201 (June) 1935.

A case of heat urticaria is described in which the wheals were caused by the application of any type of warmth. These outbreaks of urticaria were associated with increase of body temperature as well as with increase of the local temperature. Passive transfer of the sensitivity was unsuccessful. By gradually increasing the applications of heat, desensitization was accomplished.

ARE THERE SPECIFIC ANTIBODIES IN THE URINE OF SYPHILITIC PATIENTS? THOMAS PREININGER, Dermat. Ztschr. **72**:10 (Aug.) 1935.

Preininger reports that he was unable to demonstrate antibodies in the urine of patients with syphilis.

SYPHILITIC CHANCRE IN A URETHRA DUPLEX. W. KOCH, Dermat. Ztschr. **72**:15 (Aug.) 1935.

Koch reports the occurrence of a urethral chancre in a patient with a double urethra.

TAUSSIG, San Francisco.

ERYSIPELAS IN YOUNG CHILDREN. E. GORTER, Maandschr. v. kindergeneesk. **4**:51 (Nov.) 1934.

Experience with twenty-six children, of whom only five were 6 years or older, has taught Gorter that it is advisable to treat erysipelas in young children with ultraviolet radiation from a lamp which also sends out short waves. The lamp should have its maximum intensity at the beginning of the radiation, and not only the affected skin but also part of the skin around the eruption should be irradiated. The dose should be about one and one-half times the erythema dose, which must be determined for the sound skin before the beginning of the treatment, as there are great individual variations.

A NEW CATAPHORETIC SEROLOGIC REACTION FOR SYPHILIS TESTED ON 1,147 SERUMS. L. J. DELBAERE, Nederl. tijdschr. v. geneesk. **78**:48 (Dec. 1) 1934.

A serologic reaction is described, based on the lowered electrical charge carried by particles of antigen suspended in the serum of a person with syphilis. The amount of this decrease of charge is measured by means of the cataphoretic rate of displacement of these particles, which is compared with that of a lipoid dye that is simultaneously suspended in the serum.

By means of the results obtained with this reaction in tests on 1,147 serums, which were at the same time submitted to the Wassermann test and to two of the usual flocculation tests, it has been proved that this reaction is more sensitive than the very sensitive Müller reaction, and there is no reason to doubt its specificity.

VAN CREVELD, Amsterdam, Netherlands. [AM. J. DIS. CHILD.]

# Society Transactions

## CHICAGO DERMATOLOGICAL SOCIETY

MAX S. WIEN, M.D., *Secretary*

*Regular Meeting, Nov. 20, 1935*

WILLIAM ALLEN PUSEY, M.D., *President, Presiding*

A CASE FOR DIAGNOSIS. Presented by DR. E. P. ZEISLER.

This woman, aged 25, has an enlargement of the index finger of the left hand which has been present for fifteen years. Numerous firm subcutaneous nodes can be palpated on the flexor surface, and the tendon sheath is palpable as a firm cord.

Histologic examination showed marked hyperkeratosis and numerous enlarged nerve endings in the corium. No tumor cells and no xanthoma cells were visible with stains for fat.

DISCUSSION

DR. S. W. BECKER: I thought I saw some hyaline degeneration in the section of the nerve itself, not a neuroma in the sense of nerve proliferation, but indication that the nerves were definitely degenerated, and that might account for the peculiar whitish appearance of the lesions on the ball of the finger. There are swelling, hyaline change and a little fibrosis in the nerve endings but no such changes as occur in real neuromas.

DR. O. H. FOERSTER, Milwaukee: I recently saw a patient who had a history indicating that the tips of three fingers on each hand at one time were swollen and indurated and, furthermore, that the tendon sheaths of the fingers were stiffened. Several years later, when my co-workers and I saw her, we found atrophy and calcareous nodules in the tips of the fingers; the flexor tendons were shortened, and the picture was that of a sclerodermatous process. That might be a suggestion for diagnosis in Dr. Zeisler's case. There was no opportunity for biopsy in the case to which I referred.

DR. E. P. ZEISLER: I think that the disorder is some rare condition involving the tendon sheaths and causing hypertrophy of the finger; it is evidently not xanthoma or tuberculosis. The patient is in good health, and the lesion is not painful. I thought it might be a neuroma.

A CASE FOR DIAGNOSIS. Presented by DR. OLIVER S. ORMSBY.

This man, aged 65, presents an eruption of three months' duration. He has had psoriasis for twenty years, for which he has received no treatment. When this new eruption appeared there were patches of psoriasis on the extensor surface of the right forearm and over the lower portion of the spine and one small patch on the left side of the abdomen. The new eruption was rather generalized, affecting the extremities and the trunk, and was accompanied by marked pruritus.

At the time of the first examination, on Oct. 23, 1935, numerous nummular and larger bluish-red plaques were noted. The margins of several were raised and infiltrated. Irregularly scattered bullae were present, particularly on the thighs and forearms. During the month in which the patient has been under observation no new lesions other than bullae have developed, but the bullae are much more numerous than they were in the beginning.

Today, over the covered portions of the body there are numerous circular and polycyclic lesions from the size of a pea to that of a palm with raised, bluish-red

borders. Some of the patches have normal skin in the center; others present a yellowish-brown hue. There are numerous bullae of variable size on the margins of the lesions. Over the sacrum and extensor surface of the right forearm there are ordinary patches of psoriasis.

The differential leukocyte count gave the following results: polymorphonuclear leukocytes, 50 per cent; lymphocytes, 31 per cent; eosinophils, 9 per cent; monocytes, 10 per cent.

DISCUSSION

Dr. Udo J. Wile, Ann Arbor, Mich.: My impression is that there are two separate conditions, although it would be simpler if one could explain the bullae in the old lesions of psoriasis on the basis of the psoriasis. I do not think that that is altogether possible. I wonder whether this patient is taking something, such as iodine, which produces the bullous formation or whether this case is an instance of Duhring's disease developing in a patient with psoriasis. I think that in view of the serpiginous outline of the lesions on the thigh which are not scaling, a diagnosis of some form of bullous erythema in the course of psoriasis offers the most satisfastory solution.

Dr. Edward A. Skolnik: I saw this patient two months ago, and the physician who referred him stated that physical examination and studies of the urine and of the blood gave essentially negative results. The chief complaint at that time was of the severe psoriasis. Examination revealed the psoriasis and a generalized papular eruption, with circinate and bluish discolored plaques on the extensor and flexor surfaces of the forearms. There were no bullae at that time, but about a week later those lesions appeared. I have observed the patient on several occasions, and there has been practically no change except that a few new lesions have appeared. When I saw the patient I thought that the disorder was bullous erythema multiforme, and it now looks practically the same as it did then.

Dr. Clark W. Finnerud: I saw this patient some time ago at my clinic, and my impression was much like that of Dr. Skolnik. The patient had psoriasis and, I thought, also erythema multiforme rather than dermatitis herpetiformis. Later, when I saw him again with Dr. Ormsby on one occasion, there were some areas suggestive of mycosis fungoides, and at that time most of the edematous-looking plaques and bullae had disappeared, but there were some discoid and crescentic, scaling, well infiltrated plaques which were different from his psoriatic lesions.

Dr. Oliver S. Ormsby: The amount of infiltration in the plaques has diminished in the last month, and the number of bullae has increased.

Dr. O. H. Foerster, Milwaukee: The clinical picture on the thighs is that of dermatitis herpetiformis. When one takes into account the changes that have been described as taking place in the eruption, the history that the man has taken liquid petrolatum every day and the dusky appearance of the violaceous border of several of the lesions, I think one must consider phenolphthalein as a possible cause for the condition.

Dr. William Allen Pusey: I think Dr. Wile summarized the case very well. I believe one has to forget that the patient has psoriasis. He has a toxic erythema, but this is one of the cases in which one cannot tell whether the disorder is toxic erythema or a toxic erythema and Duhring's disease.

Dr. Oliver S. Ormsby: I think the discussion is very pertinent, and I believe that the condition will be proved to be bullous erythema multiforme or Duhring's dermatitis herpetiformis. I think that one cannot make a definite diagnosis at present.

Lymphoblastoma (Leukaemia Cutis). Presented by Dr. Oliver S. Ormsby.

This man, aged 65, presents a cutaneous disorder of nine months' duration, which began with marked pruritus, followed by generalized erythema and pigmentation.

The entire skin is a deep mahogany brown, the pigmentation being deepest on the trunk. Moderate adenopathy is present. There is no evidence of scaling or traumatic dermatitis from scratching. The patient's general health is good.

The leukocytes numbered 18,000 per cubic millimeter, and the results of the differential count were as follows: polymorphonuclears, 25 per cent; lymphocytes, 73 per cent, and transitional cells, 2 per cent.

DISCUSSION

DR. UDO J. WILE, Ann Arbor, Mich.: I was much interested in seeing this patient, whose condition, I think, can hardly be diagnosed unless the blood is examined. There is little that I can add to the discussion on this subject, which seems to be growing more extensive all the time, except to express my opinion that there are two distinctly different types of lymphoblastoma as seen in the skin. The first type is illustrated by Dr. Ormsby's case and consists of a primary condition of the blood with secondary cutaneous manifestations. Cases of this type comprise for the most part those in which a dermatologist sees the patient after an internist has seen him or in which the patient consults a dermatologist because of cutaneous manifestations such as leukemic nodules or other lymphoblastomatous cutaneous lesions. Under this heading are the well known dermatoses seen in association with lymphosarcoma and Hodgkin's disease.

On the other hand, there are cases in which, I believe, a lymphoblastomatous change in the skin as well as in the blood and lymph glands is secondary to certain primary cutaneous insults. This group, I believe, is even larger than that of cases of the essential or primary condition in the blood. In this group one finds cases of ordinary cutaneous disease, such as occasional cases of psoriasis and cases of external dermatitis, in which the disorder becomes universal, leading to exfoliative dermatitis, and during the course of this condition the lymph glands and not infrequently the blood become involved, and the lymphoblastomatous changes take place in the skin itself.

I have repeatedly seen the picture develop in this way, and I am convinced that one can definitely separate these two groups of cases. The cases of primary or essential lymphoblastoma are usually fatal. Recovery, however, may occasionally take place in the cases of secondary lymphoblastoma, and remission is much more common in them.

DR. OLIVER S. ORMSBY: This is the fourth patient with identical signs whom I have presented. My co-workers and I have been able to make a probable diagnosis before examining the blood. One of the patients suffered intensely, and more than eighteen months elapsed before any changes in the blood occurred. Then extensive adenopathy developed, and the patient died within a year of leukemic changes in the blood and erythroderma. I think it is important that one is able to recognize such an important disease merely by the appearance on the skin of a particular type of erythema.

A CASE FOR DIAGNOSIS (KAPOSI'S SARCOMA?). Presented by DR. THEODORE CORNBLEET.

A. L., a man aged 67, first noticed an eruption on his right leg eight years ago. The eruption increased slowly in extent, but in the last two months there has been more rapid extension.

At present the eruption consists of slate-gray to red nodules and large nodes, the largest of which is the size of a small walnut; practically all the lesions remain intact, and some of them are covered with a scaling crust. They are somewhat firm, indurated and sharply elevated and are accompanied by no subjective symptoms. No lesions have appeared on the palms or soles, except for one that formed two weeks ago on the plantar surface of the right great toe and was quite painful for several days. The patient's health has been good, and a general examination revealed no abnormalities.

Examination of the blood showed a number of young forms among the white cells, but their total number and differentiation were within normal limits.

This man has been treated by experienced dermatologists and has attended a number of well known clinics in the country. Reports from several of them showed a leaning toward a diagnosis of Kaposi's sarcoma. He is presented because a section made by some one else was examined at Dr. Cornbleet's request by Dr. Richard Jaffé, pathologist of the Cook County Hospital. Dr. Jaffé said that as far as he could see on the one section and with the reservations enforced by the lack of opportunity for making special stains (no tissue being available) the disorder was a leiomyoma. He thought that the part which had been regarded as whorls of connective tissue was in reality smooth muscle, and he advised that this diagnosis be corroborated by special stains. The one slide available showed neither areas of hemorrhage nor pigmentation. There was, however, new formation of blood vessels.

DISCUSSION

Dr. Clark W. Finnerud: I think that the disorder is Kaposi's sarcoma. The clinical appearance of the lesions is characteristic, even down to the warty appearance at the sides of the dorsa of the toes. As to the nuclei in the cells which resemble myoma, I have seen nuclei of that size, shape and situation in purely fibrous hypertrophy.

Dr. Ruben Nomland: In spite of the apparent lack of pigmentation in the microscopic section, the architecture—the intertwining of cells of the elongated type—is typical of Kaposi's sarcoma. I think that perhaps special staining of other portions of the lesion for iron would demonstrate the presence of hemosiderin.

Dr. David Lieberthal: The disorder is clinically typical of Kaposi's sarcoma, especially the lesions on the distal parts of the lower extremities. As regards the microscopic diagnosis, I should say that it is too much to ask of any one to make it after a hurried glance at the sections. But I am certain that if the sections were studied carefully the histologic picture would support the clinical diagnosis of Kaposi's sarcoma.

Dr. James H. Mitchell: In the patient I have presented here on two occasions with lesions on the hands a number of additional lesions have developed on the feet and recently one lesion has appeared in the mouth. The lesion in the mouth is clinically the same as the other lesions, being vascular and tender. I should like to know whether any of the members has seen an early lesion of Kaposi's sarcoma in the mucous membrane of the mouth.

Dr. Theodore Cornbleet: I am grateful for the discussion. I have not had an opportunity to take a specimen for biopsy as yet, but I hope to obtain material from one of the nodules and use some special stains; this should be done in order to rule out the possibility of the lesions, being fibrous tumors or muscle tumors. I shall use Van Gieson's stain and blue stains as well.

Dr. William Allen Pusey: I am willing to make a positive diagnosis of Kaposi's sarcoma without waiting for further examination.

A Case for Diagnosis. Presented by Dr. F. E. Senear, Dr. W. K. Ford, and Dr. Marcus R. Caro.

L. S., a woman aged 36, presents several pea-sized dull red solid nodules on the right ear; they are located chiefly on the concha and anthelix, and one extends into the oral canal. The lesions have been present for about two years; they are symptomless but occasionally become crusted after scratching. Several nodules have been removed by actual cautery.

The Kahn reaction was negative. Examination of the blood showed 75 per cent hemoglobin, and 10,200 leukocytes, with 67 per cent polymorphonuclear neutrophils, 25 per cent lymphocytes, 6 per cent monocytes and 2 per cent eosinophils. Urinalysis gave negative results.

Histologic examination revealed acanthosis of the epidermis except for a small central ulcer. In the center of the lesion the corium contained many dilated blood vessels, each surrounded by a thick, edematous fibrous mantle. At the sides the vessels were less markedly dilated and were surrounded by densely packed mantles of histiocytes, lymphocytes and plasma cells. Stains for tubercle bacilli and fat gave negative results.

DISCUSSION

DR. RUBEN NOMLAND: I have had the opportunity of doing two biopsies on lesions that were clinically what is called solid edema, that is, recurrent attacks of streptococcic infection, and one of those disorders involved the ear. The section from that lesion was a duplicate of the section shown. There were dilatation of the lymph spaces and a reaction of tuberculid type. I think this could be the result of a simple inflammatory reaction rather than of some specific granuloma.

DR. WILLIAM ALLEN PUSEY: That seems to me to be a logical conception of the situation; but how does Dr. Nomland account for the persistence of this inflammatory process and the edema?

DR. RUBEN NOMLAND: In cases of similar disorders there has been a history of recurrent attacks of cellulitis, and the lesions presented the same clinical and microscopic picture. I think the disorder need not be classified as specific.

DR. MARCUS R. CARO: I agree with Dr. Nomland's interpretation of the histologic observations. All I could make out was simple inflammatory reaction. The clinical history does not include any attacks of acute inflammation, which ordinarily would be present with recurrent cellulitis; therefore we still leave the diagnosis open.

A CASE FOR DIAGNOSIS (SENSITIZATION PLUS INFECTIOUS DERMATITIS?). Presented by DR. CLARK W. FINNERUD.

This man, aged 31, a milling machine operator, presents a mildly pruritic eruption of the dorsa of the hands, left cubital fossa, flexor surfaces of the forearms, elbows, upper lip, sides and front of the neck and right leg, of three weeks' duration. There is no history of previous cutaneous disorder. The general history, except for a gastric ulcer in recent years, is irrelevant. The patient states that for the past eighteen months he has been working at a machine which splashed considerable oil of cloudy, almost milky, appearance.

The patient presents a scaling, bluish-red eruption almost diffusely involving the dorsal surface of the hands and a somewhat lichenoid discrete papular involvement of the flexor surface of the forearms. A similar, but more diffuse and partially linear, papular eruption is present in the left cubital fossa and on the front and sides of the neck. There is a mild erythematous, scaling dermatitis on the face, and the upper lip is the site of a sharply demarcated area of crusting dermatitis, sycosiform in appearance. A psoriasiform plaque is present on each elbow, and an area of crusting dermatitis the size of a hand or larger, on the front of the right leg. There is a patchy erythematous scaling dermatitis at the base of the penis and in the pubic region.

The Wassermann reaction of the blood was negative. The patient has not obtained a sample of the oil for patch testing.

DISCUSSION

DR. CLARK W. FINNERUD: Only today I noticed that there is intertriginous involvement of the first interspace of the right foot, but I think that this suggestive mycotic element does not account for the entire picture.

DR. HOWARD J. PARKHURST, Toledo, Ohio: I wonder whether examinations for fungi have been made. It impressed me as perhaps more likely, if any of the disorder is due to fungi, that part of it might be a secondary toxic manifestation.

DR. LESTER M. WIEDER, Milwaukee: This patient also has lesions of the eyelids, which, he told me, have been present for six months, apparently preceding

the other lesions. Much of the disorder is intertriginous, except for the dorsal surface of the hands, elbows and shins. He stated that the lesion of the right shin began as a patch which extended peripherally. I believe that the disorder is either of mycotic or of bacterial—probably of mycotic—etiology and that some of the smaller papular lesions may be "ids" or toxic lesions. On the shoulders and in the axillary folds there are numerous scaling follicular papules, which, I thought, might be toxic lesions of this type. I think that a microscopic examination should be made of the material from lesions on the foot, and an intracutaneous injection for diagnostic purpose might be tried also.

DR. CLARK W. FINNERUD: I appreciate the suggestion that this might well be an infection with Monilia, but it is a little hard for me to conceive how that might account for the lesions on the elbow and for those on the face and neck. The eruption in the genital area is practically limited to the pubic region and the groin. I shall certainly try to demonstrate fungi, at least between the toes and in the crotch.

NOTE.—Cultures and fresh preparations in potassium hydroxide from all the involved areas gave negative results for yeast and yeastlike fungi.

A CASE FOR DIAGNOSIS. Presented by DR. MARCUS R. CARO.

P. P., a woman aged 33, is presented by courtesy of the Mandel Clinic. She states that lesions appeared on her neck about six years ago. Since then the eruption has gradually extended, involving the entire neck, the upper part of the chest and, to a lesser extent, the loins. Pruritus is severe in warm weather, especially at night.

The eruption consists of many closely aggregated papules the size of a pinhead and slightly larger. The lesions are chiefly flat, but a few are globular; they are round or angular and shiny and vary in color from flesh color to slightly more reddish. There is no enlargement of the lymph glands. The patient states that her sister has had a similar eruption for several years.

Examination of the blood gave the following results: The Wassermann reaction was negative; the hemoglobin content was 80 per cent; the leukocytes numbered 13,000 per cubic millimeter, and the differential count showed 53 per cent polymorphonuclear neutrophils, 44 per cent lymphocytes, 1 per cent monocytes and 2 per cent basophils. Urinalysis gave negative results.

Histologic examination of a biopsy specimen taken from the neck showed a dermal papule over which the epidermis was slightly flattened. In the center of the papule just beneath the epidermis the connective tissue was diffusely infiltrated by histiocytes, lymphocytes and free red corpuscles. Below this zone and extending deeply into the corium lay many sharply circumscribed, densely packed cellular nests. In each of these the walls of the blood vessels showed proliferation, amounting even to complete obliteration of the lumen. Within the walls and about the vessels lay concentric layers of histiocytes and lymphocytes, with occasional free red corpuscles, all densely packed together but separated by remaining fine strands of collagen fibers. Many large brown chromatophores were present within these nests and in the intervening corium. Staining of sections for tubercle bacilli, amyloid and fat gave negative results.

CLUTTON'S JOINT, ASYMMETRICAL (HYDRARTHROSIS). Presented by DR. M. DORNE and DR. S. J. ZAKON (by invitation).

Miss D., aged 24, American of Polish descent, went to the clinic of the Mount Sinai Hospital on Sept. 9, 1935, because of pain in the right eye. Examination by the ophthalmologist disclosed acute interstitial keratitis. The Wassermann reaction of the blood was positive, and the patient was referred to the clinic for syphilitic patients. Examination in that department on September 25 revealed interstitial keratitis affecting the right eye, Hutchinson's teeth and swelling of the left knee, which was twice the size of the right, the swelling extending into the

soft tissues of the thigh and leg. The patient stated that the swelling had been present for seven months before the appearance of the interstitial keratitis but that it was painless and did not interfere with movement. Roentgenograms showed no involvement of the bones. The serologic reaction of the blood of the patient's mother was strongly positive.

The patient was given bismuth subsalicylate, and after three injections there was complete disappearance of the swelling in the knee joint.

DISCUSSION

DR. S. J. ZAKON (by invitation): The object in presenting the patient was to show that swelling of the knee joint in patients with Clutton's hydrarthrosis is not necessarily symmetrical. Klauder (Klauder, Joseph V., and Robertson, Harold F.: *J. A. M. A.* **103**:236 [July 28] 1934) stated that this condition does not respond readily to treatment, but in this patient it responded well to two or three mild treatments.

ERYTHEMA ANNULARE CENTRIFÚGUM. Presented by DR. E. P. ZEISLER and DR. MARCUS R. CARO.

L. L., a girl aged 5 years, had a small ringlike lesion on the back of the left thigh about eight months ago. The lesion was treated with carbon dioxide snow and nearly disappeared. Recently it has been growing larger, and it shows activity in the slightly elevated, dull reddish border.

DISCUSSION

DR. FREDERICK R. SCHMIDT: Recently Dr. Ormsby discussed four types of atypical erythemas. The fourth type, I think, is called erythema gyratum simplex. My impression is that this lesion is not one of erythema annulare centrifugum but that it fits in better with a disorder I saw described as erythema gyratum simplex. I cannot remember the name of the author who described it.

DR. MARCUS R. CARO: I believe this is just a form of localized toxic erythema in annular formation.

A CASE FOR DIAGNOSIS (PERSISTENT SYMMETRICAL ERYTHEMATOUS EDEMA OF THE FACE). Presented by DR. JAMES H. MITCHELL.

This woman, aged 38, was first seen on Aug. 28, 1935, at which time the condition had existed for six months. She states that it began as a small area of erythema, which has gradually enlarged, and that the eruption was accompanied by little, if any, sensation. There was marked variation in the erythema and some in the edema. Definition had remained constant. The patient was seen a few times at the Central Free Dispensary but has refused to return because a vesicular eruption appeared on her legs after she received an injection of sodium thiosulfate. She admits that before this vesicular eruption appeared she had been walking in the woods.

The patient was referred by Dr. Nelson Percy, who reported that a complete physical examination had revealed nothing abnormal. Since the patient came under observation she has been given local treatment with a soothing ointment, one treatment with roentgen rays and one injection of 0.025 Gm. of gold and sodium thiosulfate, followed by a second injection of 0.05 Gm. with no benefit.

Examination of the blood showed 4,760,000 erythrocytes and 9,400 leukocytes. The blood pressure was 130 systolic and 34 diastolic. Examination of the urine showed an acid reaction and no albumin and sugar; microscopic examination showed from 4 to 8 pus cells per field, a few blood cells and no casts.

DISCUSSION

DR. CLARK W. FINNERUD: I treated a patient with a like condition at the time gold and sodium thiosulfate was first used. I thought the disorder was lupus

erythematosus, and in the beginning I used a few injections of sodium thiosulfate and then changed to gold and sodium thiosulfate therapy. A severe reaction developed; the patient was in bed with a high fever for several weeks, and the eruption became generalized. Subsequently it cleared entirely with local application of mild preparations and intravenous administration of sodium thiosulfate.

Dr. E. P. Zeisler: The patient presents the clinical appearance of what Kaposi called erysipelas perstans faciei. In the majority of cases the condition turned out to be acute lupus erythematosus.

Dr. David Lieberthal: I am strongly inclined to support the diagnosis of lupus erythematosus. And in connection with the mention of treatment with a gold preparation I wish to call attention to the sodium gold double salt of an amino-arylphosphinic acid. I have been using this preparation in preference to other gold compounds. It has given me good results, and I have not seen any serious reactions from it.

Dr. William Allen Pusey: The disorder seems to me a beginning of solid edema of the face. There are persistent lymphangitis and edema that persists at times. I believe the condition will gradually become worse.

Dr. James H. Mitchell: There are several reasons why I have withheld treatment. In the first place, the patient showed unmistakable signs of making trouble for the dispensary. I kept her pacified as well as I could. Another reason has been that she has a tendency to make trouble for me, and I wished to observe her and see how the disorder would progress. I thought there was a possibility of its becoming lupus erythematosus. I remember a patient in Dr. Ormsby's practice with a somewhat similar disorder which later changed to lupus erythematosus. The obvious thing is that there is some focal infection about the face. The physician who referred the patient to me examined her thoroughly and found nothing abnormal about the head or neck. Another possibility is the Libman-Sachs syndrome, but there is no sign of that. I gave two very small doses of gold and sodium thiosulfate, and the patient complained bitterly of the reaction. I did not see it, but she told me about it by telephone.

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Max S. Wien, M.D., *Secretary*

*Regular Meeting, Dec. 18, 1935*

William Allen Pusey, M.D., *President, Presiding*

A Case for Diagnosis. Presented by Dr. E. A. Oliver.

This man, aged 35, was first seen by me on Oct. 18, 1935, because of a large indurated lesion on the lower portion of the back just to the left of the vertebral column. The patient states that his wife first noticed this lesion about two and a half years ago. There was no pain or pruritus and the patient was conscious of the swelling only when the back of his office chair rubbed against it. By wearing a large pad over the lesion he was able to protect it.

There is an indurated swelling 3 inches (7.5 cm.) long by 1½ inches (3.8 cm.) wide just to the left of the vertebral column in the lower portion of the back. The lesion is red and nodular.

DISCUSSION

Dr. Udo J. Wile, Ann Arbor, Mich.: I think this lesion has many of the characteristics of a metastasis from a malignant neoplasm. It reminds me of the large plaques in the skin that occasionally follow scirrhous carcinoma of the breast. This possibility, of course, could be easily substantiated by a biopsy, and I assume that one will be made.

Dr. Ruben Nomland: A specimen for biopsy was obtained, but unfortunately the tissue was not excised quite deep enough. Most of that which was obtained was made up of the connective tissue capsule about the tumor. However, in several places there were cells which looked like foam cells that had contained a great deal of lipoid material. I believe that the lesion is a tumor of the chromaffin tissue, probably a hypernephroma.

Dr. Hamilton Montgomery, Rochester, Minn.: I did not see enough in the section presented to enable one to make a diagnosis. There were some foam cells present but not enough other features to suggest a hypernephroma. The section does not extend deeply enough. I believe that it would be much better to take another specimen for biopsy than to hazard a pathologic diagnosis on the basis of these inconclusive observations.

Dr. William Allen Pusey: I thought, as does Dr. Wile, that the tumor was probably a metastatic process, but I did not see the resemblance to carcinoma that he saw.

Dr. E. A. Oliver: I believe, as Dr. Wile does, that the lesion is a metastatic tumor of some type. It has been growing very slowly for two and a half years; hence it cannot be highly malignant. When Dr. Nomland suggested that a portion of it resembled hypernephroma tissue, I talked to several urologists and they told me that such tumors are not uncommon. The patient has promised to return to allow me to get a deeper specimen for biopsy. All that has been done so far is a complete urinalysis and a Wassermann test, both of which gave negative results. I shall try to present the patient in January with a complete diagnosis.

Fixed Eruption of Erythema Multiforme Type, Apparently Not Due to Ingestion of a Drug. Presented by Dr. S. W. Becker.

This woman, aged 24, married, was first seen in April 1934. At that time she stated that an eruption had appeared six times since September of the preceding year, each attack usually lasting about two weeks. In the beginning the lesions were limited to the lips and genitalia, but in subsequent attacks they involved the hands. The eruption consisted of bullous lesions located about the lips, mouth and genitalia, with erythematous plaques on the hands and arms. The patient stated that the identical regions were involved in each attack and that one lesion on the left side of the forehead was always present. Subsequent attacks became increasingly severe, and the condition was gradually extending.

Blood serum was sent to Dr. Welsh at the Mayo Clinic; it showed a negative reaction with antigen of erythema multiforme and a positive reaction with antigen of pemphigus.

The present attack began two weeks ago, and the lesions are now involuting.

Careful questioning of the patient regarding ingestion of drugs revealed nothing significant. No foci of infection could be found. Examination in the gynecological clinic showed slight mucoid discharge but no pelvic abnormality. The Kolmer and Kahn tests of the blood serum and examination of the urine gave negative results. The blood pressure was 120 systolic and 64 diastolic.

DISCUSSION

Dr E. A. Oliver: I saw this patient about a year ago in my office, and at that time I thought that the disorder was a severe multiform erythema. The lesions were present principally on the mucous membrane. The mouth and throat were full of lesions, and there were a number about the vulva and vagina. When I first saw the patient she was much excited because she had just come from the office of a physician who had made a diagnosis of secondary syphilis. I advised her to have all foci of infection removed, and today I learned that she had her tonsils removed, without benefit.

Dr. Udo J. Wile, Ann Arbor, Mich.: In reference to the difficulty in eliminating drugs as sources of fixed eruptions, I do not know how new this is to the members, but there are one or two substances closely related to phenolphthalein

that are not taken as drugs but that, as I have observed in the last six months, have produced changes analogous to those caused by phenolphthalein, particularly in the genitalia and mouth. One of these is erythrosin, which is largely used in coloring dentifrices and is also used as a coloring and flavoring, in the form of grenadine. All these syrups, so far as I know, are not made with erythrosin, but in one of the cases I investigated no pink substance other than the grenadine syrup had been ingested, and it was colored with this material. I am also told that bakers are using this erythrosin as a coloring material for icings, and it is not at all impossible that many of the fixed eruptions may also be due to this preparation.

DR. S. W. BECKER: I questioned the patient carefully, and she stated that she is certain that in each attack each lesion that was present in the previous attack has reappeared in the next attack but that each attack has been more severe than the preceding one and that the lesions have increased in number. She has one lesion on the forehead which, she states, has been present in each attack.

DR. OLIVER S. ORMSBY: It might be of some interest in this connection to refer to a patient with multiform erythema whom I saw yesterday. That patient had recurrent attacks in which the lesions were for the most part limited to the mouth. There had been some lesions on the extremities, but they were not like an eruption due to phenolphthalein. The patient was seen some time ago by Dr. Wile, when the lesions presented the appearance of an eruption due to phenolphthalein so he tested the patient with that drug, and this was followed by an exacerbation. Since that time the progress of the eruption has been such that it seems that phenolphthalein could be ruled out, unless it has been taken in some of the other forms mentioned today. One must recognize that erythema multiforme presents a multiform group of symptoms which sometimes simulate one disease and sometimes another. I do not know any instance in which this was demonstrated more plainly than in the case of the patient I presented last month and showed photographs of today. It is known that patients with erythema multiforme present several types of lesions and that sometimes a distinction between erythema multiforme, dermatitis herpetiformis and pemphigus is difficult if not impossible.

DR. S. W. BECKER: I appreciate the information Dr. Wile has given, for this is the third patient who has stated that in each attack the same spots have been involved with the same type of lesion. The first patient was observed through two acute attacks. During the first she was questioned carefully and mentioned the possibility of medicaments' being the cause of the eruption. When she returned at the time of the next attack the most careful questioning elicited no history of ingestion of drugs. The second patient had been seen by two other dermatologists, both of whom had questioned her regarding the ingestion of drugs. That patient was very intelligent, and my co-workers and I tried to elicit a history of ingestion of drugs, but we were entirely unable to obtain one. When the patient who is presented was first seen, in 1934, we made a diagnosis of erythema multiforme, but when she returned about a week ago and stated that more lesions had appeared in some places we went carefully into the history but could get no record of ingestion of drugs. There is an article on fixed erythema that is of interest (Wohlstein, E., and Schmidt, L.: Ueber ein fixes Erythem, *Dermat. Wchnschr.* **89**:1766, 1929). The authors stated that up to the time the article was written no one had reported a fixed erythema which was not due to drugs, but they reported the case of a man, aged 48, who had this type of eruption. After all, one realizes that the mechanism of the eruptions due to drugs is not known. The skin itself is not necessarily sensitive to the drug, as has been shown by various scratch and intradermal tests; hence there is no reason why a patient should not exhibit a fixed eruption due to a toxic manifestation from another source. I shall question this patient further regarding the possible use of these dye stuffs and see whether any enlightenment can be obtained.

DR. M. H. EBERT: I think it would be interesting to get a photograph of the patient during an acute attack in order to have a basis for comparison with future attacks.

DR. JAMES H. MITCHELL: MacKee and Wise described multiforme erythema of the fixed type. (*J. Cutan. Dis.* **36**:190, 1918). Dr. Ormsby may remember a patient seen in the dispensary several years ago who was tested for every substance one could think of but reacted to none. Photographs were taken of that patient. He had a perfectly classic fixed eruption with thousands of lesions. The photograph was in the record and could be referred to at each attack. The spots never varied in size, number or location.

DR. H. RATTNER: I saw a patient of Dr. Pusey who had recurrent attacks of erythema multiforme; one attack was traced to pink toothpaste and one to pink icing on cake.

DR. OTTO H. FOERSTER, Milwaukee: Sometimes on close questioning of an intelligent patient as to the use of laxatives, one finds, as in the case of one patient who had an eruption undoubtedly due to phenolphthalein, that the patient admits using cascara but insists that he never used phenolphthalein in any form. Investigation of the cascara sagrada preparation may show that it is fortified with phenolphthalein.

DR. HAMILTON MONTGOMERY, Rochester, Minn.: In my experience it is not unusual for a druggist to put up fluid extract of cascara containing phenolphthalein without mentioning the latter on the label on the bottle.

DR. WILLIAM ALLEN PUSEY: I think it would be impossible for any one to say that he had not taken phenolphthalein in some way. It is impossible to eliminate these things with mathematical certainty.

DR. HARRY M. HEDGE: When Dr. Ganz was here and attended a meeting of the Chicago Dermatological Society at the medical school of Northwestern University I presented a patient who, as was discovered, had an eruption due to bromide. For three months at the clinic I had questioned her about the use of drugs or any medication she had been taking, and I had received negative answers each time. The only other possibility was that of a blastomycosis. I finally presented her with that diagnosis, but when at the meeting she was asked point blank "How do you sleep?" she answered: "Very poorly; I have to take a powder every night."

EPIDERMOLYSIS BULLOSA WITH HYPERTRICHOSIS AND MELANOSIS. Presented by DR. M. E. OBERMAYER.

A woman, aged 30, of Canadian and English-American descent, states that about ten years ago, during the summer, blisters developed on the area where the strap of a golf-bag touches the shoulders. From that time on similar lesions appeared every summer when she carried a golf-bag on hot days. Five years ago bullae developed on the hands, elbows and ankles after slight trauma, and this condition has recurred every year since except during the months from November to April inclusive, during which time the patient has always been free from lesions. The bullae vary in size and depth. Some have ruptured spontaneously, while the smaller and deep-seated ones have been opened by the patient "in order to relieve the pain." All the lesions left pigmentation, and those on the elbows and ankles left atrophic areas. Eight years ago the patient noticed that her skin began to darken and that the hair on the face, legs and arms began to grow. Her libido is normal. She states that she had menstrual irregularities up to the time of her marriage. So far as she knows, no member of her family has ever had a similar condition. There is no blood relationship between her parents.

Roentgen examination revealed no abnormality in the pelvis or sella turcica. A bilateral pyelogram showed normal conditions. The dextrose tolerance test gave negative results. The basal metabolic rate was —10 per cent. The blood pressure was 114 systolic and 73 diastolic. A differential count showed that the number of lymphocytes was slightly increased, the count revealing 43 per cent.

The experimental production of bullae by the application of ultraviolet radiation and friction was tried repeatedly without success on various parts of the body.

The intradermal injection of serum from the blisters with saline solution as a control also gave negative results.

The patient has been using hot wax for depilation of the face and has had no cutaneous reaction.

DISCUSSION

DR. WILLIAM ALLEN PUSEY: The patient seems to present an interesting set of symptoms (1) epidermolysis bullosa, (2) the abnormal pigmentation and (3) hypertrichosis. I regard all these conditions as anomalies of development.

DR. M. E. OBERMAYER: This case is interesting for the reason Dr. Pusey has pointed out. The almost simultaneous onset of the eruption and of melanosis and hypertrichosis is in line with Guy's suggestion that epidermolysis bullosa represents a congenital polyglandular syndrome. Interesting also is the late onset which suggests the acquired or tardy form of the disease as described by McLeod. The absence of heredity indicates that the mode of transmission in this case is not dominant but recessive, a feature which is unusual in the dystrophic type of the disease. Studies of the hematoporphyrin content of the urine will be made later.

A CASE FOR DIAGNOSIS. Presented by DR. OLIVER S. ORMSBY.

This patient was presented at the meeting of the Chicago Dermatological Society in November, and the history is given in the proceedings of that meeting (ARCH. DERMAT. & SYPH. **33**:1081 [June] 1936).

Dr. Ormsby presented photographs of this patient to show the progress of the disorder since the first presentation.

Within two or three days after presentation the lesions on the abdomen began to enlarge, and the circles covered the entire trunk. Then near the center of the old lesions other rings started and enlarged peripherally, just as they do in erythema perstans. The bullous lesions gradually disappeared, and intravenous injections of calcium cleared up the disorder. About one week ago, when the disorder had almost entirely cleared up, it was noted that the number of eosinophils had increased to 36 per cent.

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## CLEVELAND DERMATOLOGICAL SOCIETY

J. R. DRIVER, M.D., *Reporter*

*Regular Meeting, June 27, 1935*

R. E. BARNEY, M.D., *Presiding*

DESTRUCTIVE GRANULOMA OF THE NOSE. Presented by DR. W. H. CONNOR.

C. H., a man aged 42, from the service of Dr. Cole and Dr. Driver at the City Hospital, was presented before this society in March 1932 (ARCH. DERMAT. & SYPH. **26**:773 [Oct.] 1932), at which time he was admitted to the hospital because of an extragenital chancre of the chin, associated with a generalized secondary eruption on the body. At that time the patient was receiving treatment for secondary syphilis. It was noted that there was an ulcerative process involving the right ala nasi; this, according to the patient, had been progressing slowly for five or six years. Antisyphilitic treatment had no effect on the lesion of the nose. The possibility of rhinoscleroma was considered, but as the patient did not come from Austria or southern Russia there was slight probability of that disorder, and subsequently examination of smears for the bacillus of rhinoscleroma gave negative results. The involvement has become progressively worse, and the patient now presents a marked destruction of the septal and of the alar cartilage associated with ulceration. The Wassermann test of the blood is now negative,

and roentgenograms show no evidence of tuberculosis of the lungs and no destruction of the bones in the nose. Examination of smears for tubercle bacilli gave negative results. A tuberculin test was positive with a dilution of 1:10,000.

Biopsy showed a hyperplastic epithelium with enlarged, prolonged rete pegs. Nuclear elements were vesicular, and spongiosis was present. In the corium there was a localized extensive inflammatory infiltrate of round cells with considerable edema. There were many new blood vessels and fibroblasts. A pathologic diagnosis of chronic granulomatous inflammation was made.

Except for application of local antiseptics, no treatment has been given, and no improvement has been noted.

DISCUSSION

DR. JOHN E. RAUSCHKOLB: My co-workers and I have observed this patient off and on since 1932. The condition of the nose has progressed slowly. The clinical picture suggests that the lesion may be a gumma. However, as we saw the patient when he had an early stage of syphilis and as the process on his nose had been present for several years before that, we know that the lesion is not due to syphilis. Ulceration due to phosphorus might have to be considered, but the patient has never worked with phosphorus. Studies for rhinoscleroma have given negative results. We are still unable to make a diagnosis.

DR. R. E. BARNEY: If the disease were rhinoscleroma, there would hardly be so much destruction of the tissue. The lesion of rhinoscleroma is usually a hard, infiltrated, shiny plaque.

DR. J. R. DRIVER: On account of the slow progress of this disease and in view of the fact that the patient has a positive reaction to tuberculin, I am inclined to believe that the disorder is tuberculosis, a destructive form of lupus vulgaris. The histologic picture does not exclude this diagnosis. Certainly there is a granulomatous process, and evidence has shown that it is not malignant and that it is not due to syphilis.

HEREDOSYPHILIS. Presented by DR. W. H. CONNOR.

A girl, aged 4 years, from the service of Dr. Cole and Dr. Driver at the City Hospital, nine months ago began to suffer from convulsions and progressive loss of speech associated with choreiform movements. Neurologic examination revealed spasticity of the muscles and incoordination. There were no ocular symptoms, and encephalography showed no abnormalities. Examination of the spinal fluid gave negative results in all respects. The cell count was within normal limits. The Wassermann test of the blood was strongly positive. Therapy consisting of weekly injections of acetarsone for nine months was of no benefit.

A boy, aged 14, from the service of Dr. Cole and Dr. Driver at the City Hospital, presents symptoms of juvenile dementia paralytica; he has slurring speech, tremor of the hands and tongue, sluggish pupils and hyperactive reflexes. The Wassermann test of the spinal fluid gave strongly positive results in amounts as low as 0.1 cc. A test for globulin gave moderately positive results; the colloidal gold curve was of the first zone type. The Wassermann test of the blood was strongly positive. Since October 1934 the patient has received eighteen injections of typhoid vaccine for the production of fever and 94.6 Gm. of acetarsone. In addition to this he has received ten treatments with the Kettering hypotherm. No definite improvement has resulted.

A Negro boy, aged 15, from the Service of Dr. Cole and Dr. Driver at the City Hospital, presents interstitial keratitis and iritis and hydrarthrosis (Clutton's joints). The enlargement of the knee joints has been present for about one year. There is a history of repeated attacks of interstitial keratitis and iritis. The Wassermann test of the blood was strongly positive. Examination of the spinal fluid gave negative results. Five treatments in the Kettering hypotherm, totaling twenty-five hours, with a temperature ranging from 105 to 107 F., produced great improvement of the ocular condition. The hydrarthrosis of the knee joints has improved slightly.

A Negro boy, aged 15, from the service of Dr. Cole and Dr. Driver at the City Hospital, suffers from heredosyphilis, interstitial keratitis, iritis of the right eye and hydrarthrosis (Clutton's joints). Both knee joints are swollen and tender, and fluid is present in each. Both wrists are involved similarly but to a less degree. Weekly intramuscular injections of neoarsphenamine and a preparation of bismuth and internal administration of potassium iodide continued for several weeks have produced little or no improvement. After six treatments in the Kettering hypotherm the interstitial keratitis and iritis have practically disappeared, and there has been slight improvement in the symptoms in the joints. The Wassermann test of the blood was strongly positive; the spinal fluid was normal.

SYRINGOMYELIA ASSOCIATED WITH TROPHIC DESTRUCTIVE BULLAE. Presented by DR. R. E. BARNEY.

M. W., a woman aged 41, has a lesion about ½ inch (1.27 cm.) below the base of the index finger on the dorsum of the left hand, which began five years ago as a "blister." In the past three years about nine similar lesions have appeared over the left shoulder and left anterior portion of the chest. On involution all the lesions leave scars. The patient has had difficulty in using her shoulder joints for the past two years; she complains that her throat has been dry for about three years and that for the past year there has been dysphagia off and on.

The patient is rather obese and short. On the flexor aspect of the junction of the middle and the lower third of the left forearm is an area of superficial ulceration, measuring 4 by 6 cm.; it is surrounded by a dusky, erythematous border ¼ inch (0.64 cm.) wide. Here the frayed epidermis gives evidence of a preexisting bullous lesion. The ulcer proper is covered by a grayish-white exudate. On the dorsum of the left hand, left shoulder and left breast are nine scars of previous lesions from the size of a pea to that of a dollar. The largest scar involves the left breast. There is a linear scar, 4 inches (10 cm.) long, on the extensor aspect of the upper third of the left arm, resulting from an exploratory operation. There is diminution of all types of sensation, but chiefly loss of pain and temperature in the areas supplied by the lower cervical and first seven thoracic nerves on the left side only. There is no definite motor change, save a slight muscular atrophy due to disuse. The grip of the left hand is strong. The tendon reflexes of the left arm are absent. There is no muscular fibrillation. The left arm is slightly shorter than the right. In a sitting position the patient can raise the left arm only about 45 degrees.

The Wassermann test of the blood and examination of the spinal fluid gave negative results. Roentgen examination in November 1934 revealed no widening of the mediastinal shadow. The proximal end of the shaft of the humerus was about 4 inches (10 cm.) from the glenoid cavity. On the slightest muscular exertion the end of the humerus was seen to be brought up into the glenoid cavity. There was absence of the head, tuberosities and upper portion of the shaft of the left humerus. There was some calcification in the soft tissue surrounding the glenoid cavity. The margins of the glenoid and probably the neck of the scapula were also absorbed. A roentgenogram of the esophagus revealed no changes.

Slight improvement has followed roentgen therapy.

DISCUSSION

DR. H. G. MISKJIAN: I wish to ask the presenter on what basis the diagnosis of syringomyelia was made?

DR. E. W. NETHERTON: The roentgenogram showed the destructive process to be in the upper end of the humerus and revealed a sharp line, suggesting that a portion of the bone had been removed at operation. I wish to know whether this is true or whether the defect is a result of the disease. I believe that in cases of syringomyelia of as long standing as this patient's is one would expect to see more deformity, whitlows and similar lesions. I suggest the possibility of a dermatitis artefacta.

DR. R. E. BARNEY: I realize that a number of symptoms are apparently lacking in order to establish a diagnosis of syringomyelia; moreover, the unilateral distribution does not agree with that diagnosis. One would expect bilateral involvement by this time. Again, there is an absence of whitlows and of deformity of the hand. There are no erythematous areas, cyanosis, etc. The possibility of a tumor involving the posterior nerves or the posterior horn has been considered. The bullous lesions which have developed from time to time have had somewhat of the appearance of a dermatitis artefacta. However, taking all the factors into consideration, I believe that a diagnosis of syringomyelia better explains the condition.

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## BRONX DERMATOLOGICAL SOCIETY

HENRY SILVER, M.D., *Secretary*

*Regular Meeting, Nov. 27, 1935*

EUGENE F. KELLEY, M.D., *President*

FOLLICULITIS DECALVANS ET ATROPHICANS (LITTLE). Presented by DR. SAMUEL FELDMAN.

W. B., a woman aged 46, has an irrelevant history except for an attack of acute colitis in May 1935. About the end of July of the same year, she noticed that the hair of the scalp was falling out. The loss of hair became gradually worse, and when the patient was examined on Oct. 15, 1935, she presented many small areas of baldness scattered over the scalp. These bald spots were smooth, round and slightly depressed. The follicles between the affected areas were hyperkeratotic, and the hairs, when present, were surrounded by a horny collar at their base.

The patient received two roentgen exposures of ⅙ skin unit (50 roentgens) at an interval of one week; this treatment was followed by weekly ultraviolet irradiation from a quartz mercury vapor glow lamp. The keratosis became markedly diminished, but the hair continued to fall out. At present the areas of alopecia are larger; some spots are smooth and slightly atrophic while others present dilated follicles. In the occipital region there are areas of baldness showing definite perifollicular inflammation as evidenced by redness of the follicular rim.

DISCUSSION

DR. DAVID BLOOM: Because of the irregular shiny areas of alopecia with scarring, I suggest the diagnosis of healed favus.

DR. ADOLPH ROSTENBERG: The disorder is evidently a folliculitis ending in atrophy and a loss of hair. It is therefore my impression that it is folliculitis decalvans.

DR. VAN ALSTYNE H. CORNELL: I also think that the disorder is folliculitis decalvans, although the possibility of lupus erythematosus must be considered.

DR. F. E. CROSS: I think that the suggestions of Dr. Cornell are tenable. I do not agree with Dr. Bloom's diagnosis of favus in view of the fact that favus is more widespread over the scalp and that the areas of atrophy are not as irregular as in this case. Perhaps Dr. Feldman can give some information on the question of a history of favus. To me the disorder suggests pseudopelade.

DR. SAMUEL FELDMAN: I expected a difference of opinion because so few cases of this disorder have been reported and the differential diagnosis has not been sufficiently stressed. The condition that this patient presents belongs to a group which has not as yet been properly classified. The condition was described by Graham Little as folliculitis decalvans et atrophicans. I discussed this condition

under the title of lichen planus et accuminatus atrophicans (ARCH. DERMAT. & SYPH. **5**:102 [Jan.] 1922) since in most of the cases definite lesions of lichen planus were seen, and my conception is that the process is a variation of lichen planus. Little in a personal communication stated that he agrees with my view. This patient certainly has no folliculitis decalvans. There are no pustules and there have not been any at any time and there is no scarring. On the other hand, there are atrophy and horny plugs, the conditions described by Little and by me. The differences consist in the absence of lesions on the body and of lesions of lichen planus in this case. As to the question of favus, the lack of actual scarring and the presence of follicular plugs are sufficient to rule out that diagnosis. Moreover, the patient's son, a physician, definitely asserts that there is no history of favus.

HERPES ZOSTER GENERALISATUS. Presented by DR. PAUL GROSS.

This man, aged 32, married, born in Estonia, came to the clinic of the Hospital for Joint Diseases on Nov. 4, 1935, presenting typical herpes zoster on the left side of the neck. The patient received solution of posterior pituitary on that day and on the following day. On November 6 he was admitted to the hospital because of an eruption covering the entire face and the trunk down to the waist line, marked cervical adenopathy and a temperature of 102.4 F.

The original group of lesions on the left side of the neck had further developed; it had become confluent, and some lesions had become gangrenous. The eruption on the face and trunk was characterized by papules and vesicles with a bright red erythematous halo typical of lesions seen in varicella. The patient was given one injection of a 50 per cent dextrose solution intravenously and three injections of a solution of sodium iodide and was discharged on November 9. The eruption had completely healed, but the patient still complained of pain on the left side of the neck. There were scars at the site of the original zoster, but the lesions which were distributed on other parts of the body healed without scarring.

DISCUSSION

DR. ARTHUR SAYER: Does Dr. Gross wish to convey the impression that all of the lesions present are lesions of zoster and are due to the same cause? How soon after the appearance of the typical zoster did the other type of lesions develop?

DR. ADOLPH ROSTENBERG: Was this patient exposed to varicella?

DR. DAVID BLOOM: There are a number of reports in the literature about varicella's having developed in persons who came in contact with patients with zoster and vice versa. There are also reports of herpes zoster and varicella occurring in the same person. These facts indicate the probability that zoster and varicella have the same etiologic factor.

DR. PAUL GROSS: Several years ago I had occasion to observe another case of the same disorder. In that case the herpes zoster appeared in the left frontal region, and after a rise in temperature and marked cervical adenitis symmetrical lesions of zoster type appeared on both ears and on the dorsa of the hands. These cases of herpes zoster generalisatus offer one of the arguments for the identity of the virus of herpes zoster and that of varicella. In answer to Dr. Rostenberg, I wish to state that, as far as is known, this patient has not been exposed to varicella.

LICHEN PLANUS WITH UNUSUAL FEATURES. Presented by DR. LOUIS CHARGIN.

This man, aged 40, a salesman, states that his eruption first appeared on the forearms about four months ago. It soon spread to the hands, legs and genitals.

There is well marked lichen planus, somewhat subacute in type, affecting the forearms, wrists, palms, lower third of the legs, soles and genitals. On the forearms the lesions are rather large and bluish red. On the palms and soles the

eruption closely resembles keratoderma maculosum palmare et plantare (Buschke and Fischer). The lesions are isolated, scaly at the border and show a well marked central depression. Over the heels the eruption is yellowish and hyperkeratotic. The nails are markedly affected, being irregular on the surface and showing transverse ridges and flakes. There is some subungual hyperkeratosis. The mucous membranes of the cheeks are covered with a large number of lesions showing circular arrangement and segments of circles. Except for the cutaneous disorder the patient is well. He complains of some itching. The trunk is free from lesions.

DISCUSSION

DR. F. E. CROSS: This is an interesting case, especially because of the lesions on the palms. I have seen three or four patients with similar manifestations of lichen planus, but this one exhibits the most characteristic lesions. The involvement of the nails is marked. Lichen planus of the nails has been recently reported by Dr. Frank Vero (ARCH. DERMAT. & SYPH. **26**:677 [Oct.] 1932).

DR. ARTURO L. CARRIONN (by invitation): I think it is worth while to state that in Puerto Rico one sees a large number of patients with lichen planus that is atypical in its distribution. Often one sees lichen planus of the extensor surfaces, chiefly on the dorsa of the hands. In the last two years I have observed three cases of lichen planus of the hands; in one of them there were also lesions on the soles. In a particularly interesting case lesions which were not suggestive of lichen planus developed first on both palms, and it was only later, when typical lesions developed farther up on the wrists, that the diagnosis could be definitely established. It is not always necessary that the eruption be acute or generalized before one finds lesions on the palms.

DR. CHARLES A. GREENHOUSE: What therapy will be instituted in this case?

DR. LOUIS CHARGIN: In answer to Dr. Greenhouse I wish to state that in addition to administration of arsenic it will no doubt be necessary to resort to roentgen therapy, especially for the palmar and plantar lesions. If only the lesions on the palms and soles were shown, even the most experienced clinician would most likely miss the diagnosis because the lesions so closely resemble the macular disseminated keratoderma described by Buschke and Fischer.

A CASE FOR DIAGNOSIS (MELANOTIC WHITLOW? SQUAMOUS CELL EPITHELIOMA?). Presented by DR. PAUL GROSS.

This woman, aged 59, born in the United States, is presented from the Hospital for Joint Diseases. The patient states that two years ago an ironing board fell on her left foot. Since then there has been a bluish discoloration under the nail of the left big toe. About six months ago the toe increased in size and began to suppurate. The distal portion of the left big toe shows a granulating mass with a somewhat verrucous surface surrounded by a hyperkeratotic area. The distal portion of the nail is destroyed; the proximal part is raised by the tumor mass and shows a bluish discoloration of the nail bed. There is no enlargement of the inguinal glands.

Sections from the granulating tumor were taken for histologic study about six weeks ago and revealed a squamous cell epithelioma.

The patient is presented because the history and location suggest a melanotic whitlow, while the histologic picture conclusively shows a squamous cell epithelioma. The patient is also presented for therapeutic suggestions.

DISCUSSION

DR. DAVID BLOOM: I agree with the diagnosis of melanotic whitlow on clinical grounds. The treatment of choice is, I believe, surgical, and should consist of removal of the toe.

DR. ARTHUR SAYER: In view of the likely serious character of the condition I believe that it is imperative to amputate the toe. After that is done, further

pathologic studies should be made to determine the diagnosis more definitely. The loss of the toe should not deter from the indicated therapy.

DR. F. E. CROSS: As a prophylactic measure roentgen treatment should be given to the glands in the groin.

DR. PAUL GROSS: I am thankful for the suggestion. I also believe that amputation of the toe is the method of choice.

NOTE.—The toe was amputated on Dec. 4, 1935, and the section of the tumor showed melanocarcinoma, the dopa reaction being positive throughout the entire tumor mass.

DERMATITIS VEGETANS DUE TO FUNGUS. Presented by DR. SAMUEL FELDMAN.

T. F., a youth aged 19, noticed a scaling of the left foot during July 1935. He paid no attention to the condition, and about one month later pruritus of both groins and of the scrotum developed.

On Oct. 2, 1935, there were marked redness, scaling and crusting in the interspaces between the third, fourth and fifth toes of the left foot, the involvement extending to the dorsal surface. There was evidence of much scratching and irritation. In both groins and on the sides of the scrotum there were thickening and crusting.

Microscopic examination of scales from the foot revealed short chains of mycelial segments. A culture on a 4 per cent solution of dextrose agar produced a white fuzzy growth similar to that found in cultures of Epidermophyton.

The patient received six weekly roentgen irradiations of ¼ skin unit (75 roentgens) each. By the end of that treatment the lesions in the groin became less crusted. After the surface was thoroughly cleansed three oblong patches were seen, each one being made up of a conglomeration of macerated and even ulcerated papules. The condition is somewhat better at present, but it yields to treatment with great difficulty.

The biopsy made on November 17 shows that the epithelial layer is much distorted and thrown into irregular folds. There are intracellular and extracellular edema and marked parakeratosis, and the granular layer is for the most part completely lacking. There are acanthosis and elongation and distortion of the papillae, edema of the corium, vascular dilatation and perivascular round cell infiltration.

# Book Reviews

**Recent Advances in Dermatology.** By W. Noel Goldsmith. Price, $5. Pp. 522, with 8 colored plates and 50 illustrations. Philadelphia: P. Blakiston's Son & Co., Inc., 1936.

This work makes an effort which is new in dermatology; that is to bring together and consider critically the recent essential additions to the knowledge of dermatology in what is the modern period of the various subjects which it takes up and then, as far as may be, to interpret in the light of this knowledge various diseases of the skin. For example, it considers the discoveries and advances in the field of allergy which have given the present conception of that reaction and then interprets various diseases in the light of the new knowledge of allergy. The knowledge of the subjects is not merely summarized, as in the usual text-books and year-books, but the discussion covers what may be considered the evolution in the recent period in any field, whether five or twenty-five years.

The first few chapters take up the important new additions to the general knowledge of dermatology, and it is assumed in the discussions which follow that the reader is familiar with these chapters. The author states: "Conclusions drawn from evidence sifted in the earlier chapters are taken for granted later. Thus, throughout the rest of the book, a familiarity is assumed with Lewis' work on the Triple Response, dealt with in Chapter I.: disorder of Lipoid Metabolism in Chapter VI, cannot be discussed without reference to the Reticulo-endothelial System, which has therefore been made the subject of Chapter V.: an understanding of the manifestations of Cutaneous Tuberculosis (Chapter VIII) demands a careful study of the principles of Allergy, which I have tried to disentangle in Chapter VII." The scope of the whole and the method of presentation are indicated by the headings of the chapter, but these hardly indicate the interest of the contents. For example, chapter I is entitled "Blood Vessels," and in this Goldsmith considers extremely interesting new additions to the knowledge in that field. Chapter II is entitled "Glands of the Skin," and Chapter III, "Pigmentation." Both these subjects are commonplace, but with the emphasis on the new knowledge they are made extremely interesting. Other chapters are "Endocrine, Nervous and Mental Influences"; "Reticulo-Endothelial System"; "Disorders of Metabolism"; "Allergy," and "Filter-passing Viruses." There are fourteen chapters in all.

The book is an able and remarkably interesting presentation of the new knowledge of dermatology. It is something unusual in books on dermatology. It integrates the recent advancements in dermatology with the familiar knowledge of this subject, and tends to clarify the knowledge of the principles which underlie pathologic processes and thus broadens one's comprehension of the subject. It can be read universally with profit.

**Year Book of Dermatology and Syphilology, 1935.** Edited by Fred Wise and M. Sulzberger. Price, $3. Pp. 736, with 90 illustrations. Chicago: Year Book Publishers, Inc.

The reviewer is glad to call attention to this volume of the Year Book. These Year Books have for many years been furnishing valuable summaries of the progress of dermatologic knowledge, and this volume maintains the high standard. The editors have selected their material wisely and in all ways have carried out well their responsibilities. One could realize how valuable such a book is in keeping one abreast of dermatologic knowledge only if one tried to review the literature of any topic in dermatology without the aid of such a work. The book deserves circulation among all who have occasion to treat cutaneous diseases.

The editors introduced an innovation last year by beginning the volume with an article of their own on acne. They begin this year's volume with an article on eczema. The innovation seems to the reviewer of questionable desirability. These articles are not properly within the scope of the volume and are apt to be misleading on this point to the reader who takes up the book for the first time. The practice in treating acne and eczema as described in the two articles that appeared in the last two years seems hardly to represent that generally accepted in treating these two troublesome diseases and would be difficult for the practitioner to follow.

**Commoner Diseases of the Skin.** By S. William Becker. Price, $4. Pp. 283, with 83 illustrations. New York: National Medical Book Company, Inc., 1935.

This book covers well the commoner diseases of the skin. The style is didactic (which is a good feature for such a book), and the descriptions are concise and clear. Some chapters are particularly good, for example that on allergy in cutaneous diseases. The book ends with a useful formulary, which is particularly to be recommended because it confines itself to remedies or methods that are in common use and does not confuse the inexperienced practitioner with a multiplicity of little used remedies. The work is of value in furnishing an orientation of the familiar diseases of the skin. The only restriction that must be put on this recommendation is that Becker ascribes to "nervous hyperactivity and exhaustion" in the production of cutaneous diseases an importance far greater than most dermatologists accept. The new student of dermatology will get from his text an exaggerated notion of the importance of the psychogenic and neurogenic factors in diseases of the skin.

**Radium Treatment of Skin Diseases, New Growths, Diseases of the Eyes and Tonsils.** By Francis H. Williams. Price, $2. Pp. 118, with 12 illustrations. Boston: Stratford Company, 1935.

This is a personal book. Coming from Francis H. Williams, it represents the experience of one of the earliest workers in radiotherapy, with both roentgen rays and radium. Dr. Williams wrote a notable book on the subject thirty-five years ago, which was based largely on his own experience, and he has been working in the same field until the present. Unfortunately, only a few pages are devoted to diseases of the skin, but the whole is interesting to any worker in radiotherapy because of the record and wide experience of the author.

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## CORRECTION

In the review of "Emotions and Bodily Changes, A Survey of Literature on Psychosomatic Interrelationships, 1910-1933," in the April issue (ARCH. DERMAT. & SYPH. **33**:776, 1936), the pronouns "he" and "his" throughout the review should be "she" and "her."

# Directory of Dermatologic Societies *

## INTERNATIONAL

NINTH INTERNATIONAL CONGRESS OF DERMATOLOGY AND SYPHILOLOGY
American Secretary: Dr. Howard Fox, 140 E. 54th St., New York.

## FOREIGN

BRITISH ASSOCIATION OF DERMATOLOGY AND SYPHILOLOGY
(CANADIAN BRANCH)

H. A. Dixon, President, Medical Arts Bldg., Toronto, Ont.
Norman Wrong, Secretary-Treasurer, Medical Arts Bldg., Toronto, Ont.

ROYAL SOCIETY OF MEDICINE, SECTION OF DERMATOLOGY

H. W. Barber, President, 7, Devonshire Pl., London, W. 1.
Louis Forman, Secretary, 7, Devonshire Pl., London, W. 1.

## NATIONAL DERMATOLOGIC SOCIETIES

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Paul A. O'Leary, Chairman, 102 Second Ave., S.W., Rochester, Minn.
J. Bedford Shelmire, Secretary, 1719 Pacific Ave., Dallas, Texas.
Place: Atlantic City, N. J.

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOLOGY

Howard Fox, President, 140 E. 54th St., New York.
C. Guy Lane, Secretary, 416 Marlborough St., Boston.

AMERICAN DERMATOLOGICAL ASSOCIATION

C. Guy Lane, President, 416 Marlboro St., Boston.
F. D. Weidman, Secretary, University of Pennsylvania, Philadelphia.
Place: New Ocean House, Swampscott, Mass. Time: June 4-6, 1936.

## SECTIONAL DERMATOLOGIC SOCIETIES

CENTRAL STATES DERMATOLOGICAL ASSOCIATION

Samuel Goldblatt, President, 3208 Colerain Ave., Cincinnati.
Raymond G. Senour, Secretary-Treasurer, 19 W. 7th St., Cincinnati.
Place: Cleveland. Time: Dec. 5, 1936.

MISSISSIPPI VALLEY DERMATOLOGICAL SOCIETY

Samuel Goldblatt, President, 3208 Colerain Ave., Cincinnati.
Raymond G. Senour, Secretary-Treasurer, 19 W. 7th St., Cincinnati.

* Secretaries of dermatologic societies are requested to furnish the information necessary for the editor to make this list complete and to keep it up to date.

NORTHERN NEW JERSEY DERMATOLOGICAL SOCIETY

Henry J. F. Wallhauser, President, Medical Tower, Newark.
Louis J. B. LeBel, Secretary, 165 Grant Ave., Nutley.
Place: Academy of Medicine of Northern New Jersey, Newark. Time: Third Tuesday of January, March, May, September and November.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Jack W. Jones, Chairman, 384 Peachtree St., Atlanta, Ga.
Winston U. Rutledge, Secretary, 332 W. Broadway, Louisville, Ky.
Place: Baltimore. Time: Nov. 17-20, 1936.

## STATE DERMATOLOGIC SOCIETIES

CALIFORNIA MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, DERMATOLOGY AND SYPHILOLOGY SECTION

William H. Goeckerman, Chairman, 1680 N. Vine St., Hollywood.
Stanley O. Chambers, Secretary, 1210 Roosevelt Bldg., Los Angeles.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON DERMATOLOGY

Ellwood C. Weise, Chairman, 144 Golden Hill St., Bridgeport.
Edward A. Abbey, Secretary, 442 Temple St., New Haven.

FLORIDA SOCIETY OF DERMATOLOGY AND SYPHILOLOGY

J. L. Kirby-Smith, Chairman, 204 Laura St., Jacksonville.
W. M. Sams, Secretary, 310 Ingram Bldg., Miami.

LOUISIANA DERMATOLOGICAL SOCIETY

M. T. Van Studdiford, President, 912 Pere Marquette Bldg., New Orleans.
R. A. Oriol, Secretary-Treasurer, Maison Blanche Bldg., New Orleans.

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Harvey P. Towle, President, 453 Marlborough St., Boston.
Rudolf Jacoby, Secretary, 270 Commonwealth Ave., Boston.

MEDICAL SOCIETY OF THE STATE OF NEW YORK, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Louis Tulipan, Chairman, 224 E. 17th St., New York.
C. H. Peachey, Secretary, 197 S. Goodman St., Rochester.

MEDICAL SOCIETY OF THE STATE OF PENNSYLVANIA SECTION ON DERMATOLOGY

Abraham Strauss, Chairman, Medical Arts Bldg., Philadelphia.
L. G. Bembauer, Secretary, 5026 Jenkins Arcade Bldg., Pittsburgh.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON DERMATOLOGY AND SYPHILOLOGY

A. E. Schiller, Chairman, 10 Peterboro St., Detroit.
G. Warren Hyde, Secretary, 2501 W. Grand Blvd., Detroit.

MINNESOTA DERMATOLOGICAL SOCIETY

L. H. Winer, President, 78 S. Ninth St., Minneapolis.
F. W. Lynch, Secretary, Lowry Medical Arts Bldg., St. Paul.
Time: First Wednesday in October, December, February and April.

### Oklahoma State Dermatological Society

Marque O. Nelson, President, 307 Medical Arts Bldg., Tulsa.
Harry Green, Secretary, 1116 Medical Arts Bldg., Tulsa.

### Texas Dermatological Society

J. C. Michael, President, 1215 Walker Ave., Houston.
Everett R. Seale, Secretary, 1215 Walker Ave., Houston.
Place: Austin.

## LOCAL DERMATOLOGIC SOCIETIES

### Baltimore-Washington Dermatological Society

Dudley C. Smith, President, University of Virginia Hospital, Charlottesville, Va.
Francis A. Ellis, Secretary, 104 W. Madison St., Baltimore.

### Bronx Dermatological Society

Eugene F. Kelley, Chairman, 93 Radford St., Yonkers, N. Y.
Henry Silver, Secretary, 290 West End Ave., New York.

### Brooklyn Dermatological Society

Samuel Hecht, President, 679 Greene Ave., Brooklyn.
Mortimer J. Cantor, Secretary, 907 St. Marks Ave., Brooklyn.
Time: Third Monday of each month except June, July, August and September.

### Buffalo-Rochester Dermatological Society

Earl L. Eaton, President, 40 North St., Buffalo.
Richard L. Saunders, Secretary, 333 Linwood Ave., Buffalo.

### Central New York Dermatological Society

George M. Fisher, President, 264 Genesee St., Utica.
Rudolph Ruedemann Jr., Secretary-Treasurer, 214 State St., Albany.

### Chicago Dermatological Society

Max S. Wien, President, 104 S. Michigan Ave., Chicago.
Reuben Nomland, Secretary, 25 E. Washington St., Chicago.

### Cincinnati Dermatological Society

Raymond G. Senour, President, 19 W. 7th St., Cincinnati.
Leon Goldman, Secretary-Treasurer, Doctor's Bldg., Cincinnati.

### Cleveland Dermatological Society

H. G. Miskjian, President, 856 Rose Bldg., Cleveland.
E. W. Netherton, Secretary, 2020 E. 93d St., Cleveland.

### Detroit Dermatological Society

L. W. Shaffer, President, 1553 Woodward Ave., Detroit.
William G. Saunders, Secretary-Treasurer, 9203 Grand River Ave., Detroit.

### Los Angeles Dermatological Society

N. P. Anderson, Chairman, 2007 Wilshire Blvd., Los Angeles.
Thomas W. Nisbet, Secretary, 65 N. Madison Ave., Pasadena, Calif.
Time: Second Tuesday of each month, October to May, inclusive.

MANHATTAN DERMATOLOGIC SOCIETY

Paul E. Bechet, Chairman, 55 Park Ave., New York.
Mihran B. Parounagian, Secretary, 126 E. 39th St., New York.

MONTREAL DERMATOLOGICAL SOCIETY

Alberic Marin, President, 1414 Drummond St., Montreal, Canada.
Paul Poirier, Secretary, 2073 St. Denis St., Montreal, Canada.

NEW ENGLAND DERMATOLOGICAL SOCIETY

William P. Boardman, President, 388 Marlborough St., Boston.
J. Harper Blaisdell, Secretary, 45 Bay State Road, Boston.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

Max Scheer, Chairman, 509 Madison Ave., New York.
Frank C. Combes, Secretary, 80 W. 40th St., New York.

NEW YORK DERMATOLOGICAL SOCIETY

R. H. Rulison, Chairman, 114 E. 62nd St., New York.
Eugene F. Traub, Secretary, 140 E. 54th St., New York.

OMAHA DERMATOLOGICAL SOCIETY

J. A. Borghoff, Chairman, 1319 Medical Arts Bldg., Omaha.
Donald J. Wilson, Secretary, 1113 Medical Arts Bldg., Omaha.

PHILADELPHIA DERMATOLOGICAL SOCIETY

Herbert J. Smith, Chairman, 3305 N. 17th St., Philadelphia.
Vaughn C. Garner, Secretary, Greene and Coulter Sts., Philadelphia.

PITTSBURGH DERMATOLOGICAL SOCIETY

Emerson Gillespie, President, 120 Tuscarawas St., Canton, Ohio.
Joseph J. Hecht, Secretary, 506 Medical Arts Bldg., Pittsburgh.

ST. LOUIS DERMATOLOGICAL SOCIETY

A. H. Conrad, President, 3720 Washington Blvd., St. Louis.
Roy L. Kile, Secretary-Treasurer, 3720 Washington Blvd., St. Louis.
Place: Barnard Free Skin and Cancer Hospital. Time: Second Wednesday of each month.

SAN FRANCISCO DERMATOLOGICAL SOCIETY

L. R. Taussig, President, 384 Post St., San Francisco.
John M. Graves, Secretary, 909 Hyde St., San Francisco.

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